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ACUTE LARYNGOTRACHEOBRONCHITIS: TREATMENT OF THE OBSTRUCTIVE LARYNGEAL AND TRACHEOBRONCHIAL EDEMA WITH HYPERTONIC HUMAN PLASMA. A PRELIMINARY REPORT.

DR. HARRY L. BAUM, Denver.

The clinical entity laryngotracheobronchitis almost always affects young children and is characterized by rapid onset of respiratory symptoms with hoarseness, cough, fever and laryngeal stridor. The stridor progresses, sometimes rapidly, to almost complete obstruction, necessitating intervention by means of intubation or tracheotomy. The cause of the laryngeal obstruction is an inflammatory edema in the subglottic tissues, and this condition of inflammation and edema extends throughout the mucosa of the tracheobronchial tract, with marked alteration of the secretions, which become thicker and more adherent. Usually, of course, because of the looser character of the subglottic tissues, the edema in this location is more marked than it is in the tracheobronchial mucosa and submucosa, but it is an important feature of the pathological process throughout.

If the laryngeal obstruction is not relieved, the patient dies — not usually from asphyxia but from exhaustion caused by the prolonged, violent respiratory effort, with secondary myocardial failure. If the laryngeal obstruction is relieved, usually accomplished by tracheotomy, about 50 per cent of the patients die anyway from the results of obstruction lower down. The secretions become still more thickened and glue-like, and inspissated plugs of semidried secretion accumulate

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in the main bronchi. If these are not removed, the patient succumbs rapidly from asphyxia. If they are removed they often recur, and, in spite of repeated removals, obstruction of the smaller bronchi and bronchioles sometimes follows. This development is a sequel of the edema, with the added formation of pseudomembrane in the smaller air passages. It results in areas of obstructive atelectasis, and sometimes in bronchopneumonia. Death eventually supervenes from asphyxia, exhaustion and toxemia, if not from pneumonia, although toxemia is not uniformly a prominent factor in these cases. A very high mortality rate in laryngotracheobronchitis is the uniform experience, regardless of the untiring efforts of conscientious medical attendants.

It is significant that the extreme thickening of the tracheobronchial secretions, and the consequent bronchial plugging, seem invariably to follow establishment of an artificial airway and never to precede it. This is not true of the inflammation and edema, which are primary and extend into the smaller bronchi at the earliest stage of the disease. With these facts in mind, and also to avoid the hazards of tracheotomy, which are not in themselves inconsiderable, I have sought for a method of avoidance of all forms of surgical interference, where possible, and have at last found it — in the intravenous administration of hypertonic human plasma. While the use of concentrated plasma is well established for treatment of shock, burns, cerebral and nephrotic edema, and certain other conditions, so far as I have been able to discover it has never been suggested for the purpose of reducing the edema of the subglottic tissues and the tracheobronchial mucous membranes in laryngotracheobronchitis. For this purpose the results have been excellent in the small number of cases in which it has been tried up to the time of this preliminary report.

RATIONALE.

It should be clearly understood that the administration of hypertonic plasma is in no sense intended as a cure for the infection itself in laryngotracheobronchitis. There is little if any therapeutic value in the plasma, as such, because it presumably contains few, if any, specific antibodies against the infection from which the patient is suffering. It is the osmotic action of the hypertonic plasma protein introduced into the

blood stream by this method which accomplishes the object, and that object is the withdrawal into the blood stream of the fluid of edema in the interstitial tissue spaces of the subglottic area, and in the mucosa and submucosa of the tracheobronchial tree. It is the obstructive edema which is the serious factor in these cases. Without it, in most instances, the disease would yield to the defensive machinery provided by nature. But with the obstructive edema, the altered secretions and the secondary complications resulting from the instrumentation which they entail, we have a very different situation. When the edema is reduced, with the resultant improvement in the character of the secretions, the really formidable feature of the disease has been removed. Intravenous administration of hypertonic human plasma reduces the edema by abstracting fluid from the tissues, and seems to hold that fluid in the blood stream where it belongs.

PRELIMINARY VENESECTION.

If a considerable dosage of hypertonic plasma or serum is to be administered, and more especially if there is any suspicion of circulatory embarrassment, it is very desirable to withdraw a moderate amount of blood as a preliminary procedure. The amount of blood withdrawn should be three or four times the quantity of concentrated plasma to be administered, because administration of concentrated plasma increases blood volume to that extent. Of course, blood-letting itself also tends to reduce edema, and may for that reason be beneficial in these cases; however, that is not the chief reason for utilizing it. The compelling argument is that since administration of hypertonic plasma solution draws fluid from the tissues into the blood stream, to avoid congestion it seems advisable to remove a compensating amount of blood before administration of the plasma. If this is not done, and a large dose of hypertonic plasma is given, it is at least theoretically possible that an overloaded circulation might, as a result, cause an increase in the dyspnea, instead of the desired decrease.

ADMINISTRATION.

The method is extremely simple and entirely safe. In routine clinical use, dessicated (lyophile) plasma would ordinarily be restored to its normal consistency by addition of

the full amount of water evaporated in process of preparation. That is, if the dried plasma material has been derived from 250 cc. of citrated normal plasma originally, and since the remaining solids amount to about 10 per cent of the volume, approximately 225 cc. of water would be needed to restore it to its normal, isotonic state; but, in the restoration of dried plasma for the present purpose, that is, in the formation of four times concentrated hypertonic plasma, only one-fourth the amount of water is needed for addition to the dried material. Lyophile or dessicated plasma is now prepared by some serum centers, and also commercially, and is readily available at many drug and supply stores. It is usually supplied in a 250 cc. size. In practice, instead of restoring this with the full amount (225 cc.) of water, I restore it with about 60 cc., thus making (approximately) a one-fourth dilution, or a four times concentrated solution. The exact concentration is not important, so long as the plasma is all restored, without any dry lumps, and is not too thick to be passed through an intravenous needle.

If the patient is in only a moderately severe dyspneic state, it is wise to make use of the humidified oxygen tent until the plasma is prepared. If the condition is more serious, gentle laryngeal suction through the direct speculum, followed by the oxygen tent, should be utilized. If the danger is imminent, temporary direct intubation may be used, with a very small bronchoscope or smaller suction tube, being careful to avoid trauma to as great an extent as possible. If tracheotomy has already been resorted to, the intravenous treatment should be administered anyway, as it is as important to treat the bronchial edema and altered secretions as it is to relieve the laryngeal dyspnea in the first place; also, the treatment may permit early decannulation, which adds much to the patient's chances. It takes but a few minutes to prepare the plasma and to cut down on a vein (which is advisable in a small child), and it is well worth a little extra effort to endeavor to tide the patient safely over this short period in order, if at all possible, to avoid the necessity for instrumentation.

From 25 to 40 cc. of the four times concentrated plasma is usually administered, depending on the size of the child and the severity of the case. No reaction is to be expected, except

very rarely the slight febrile reaction which may follow any serum or plasma injection. In my own fairly extensive experience with human convalescent serum and plasma, such reactions have never run even as high as 1 per cent, and when they do occur they are negligible; so no preliminary skin testing is called for in these desperate cases. The cannula should be left in the vein, with stylet, if an incision has been made, for subsequent injections which can be made as required.

RESULTS.

The results of this treatment have been very encouraging. Almost before the injection has been completed the patient becomes tranquil, the respiratory obstruction is gone, or nearly so, the breathing is quiet, color is restored, and the little patient falls asleep on the table. It should be emphasized that the disappearance of obstructive symptoms does not indicate that the actual infection is in any sense cured; however, the vital respiratory function is relieved, and there remains only a relatively simple nonobstructive inflammation to be dealt with. In severe cases, of course, it is possible that the edema may return, and that the administration of the hypertonic plasma should be repeated.

In some epidemics, and in certain individual cases, the infection is more severe than in others, and pneumonia, septicemia or some other rapidly lethal complication may be a concomitant of the laryngotracheobronchitis. This, however, is another problem and not to be permitted to confuse the classic picture which is here under discussion.

OTHER USES.

Similar use of this method is applicable to many other types of laryngeal or tracheobronchial edema. They include postbronchoscopic obstructive traumatic edema; vegetal foreign body reactions; supraglottic edematous laryngitis with obstruction; subglottic obstructive laryngitis, with edema; angioneurotic edema; edema of the larynx from trauma; and tracheobronchial edema of any type, including crises of bronchial asthma.

COMMENT.

It is hoped that enthusiasm for an easy triumph over a serious disease will not lead to indiscriminate use of the pro-

posed method. If so, it will be unfairly discredited. It is definitely not applicable to any form of laryngitis or laryngotracheobronchitis but one in which obstructive edema is present. Concentrated plasma has no effect on the infection or the resultant inflammation — only on the edema; furthermore, dessicated plasma is expensive when purchased commercially and no one will care to waste it. Its use in various inflammatory conditions, without edema, in the larynx and tracheobronchial tract is not indicated, and no results should be expected.

Since the results of this treatment have proven satisfactory, and such cases are so serious and difficult, and since present methods of treatment are admittedly ineffectual, it is considered wise to make this early preliminary report. It is hoped that others, also interested in this subject, may soon make the necessary final test of impartial and extended trial, for the benefit of those little sufferers who might otherwise not have a chance to survive.

510 Republic Building.

AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be held in Chicago at the Palmer House on Oct. 6-7-8-9, 1943.

ONCOCYTOMA OF THE TRACHEA.*

DR. ARTHUR PALMER, New York.

Tumors of the trachea present certain problems of management which are illustrated by this report of an unusual tumor described by Hamperl and called by him oncocytoma.

Depending upon its size and type, tracheal tumor may cause little or considerable obstruction to the airway with attendant embarrassment of respiration. Emphysema, atelectasis, bronchiectasis and lung suppuration are the more common complications which may arise if the obstruction continues.

Complete removal of a tracheal tumor with a punch forceps, snare, surgical diathermy, or by a combination of these methods, is not always possible or practical. The bronchoscopist must consider the dangers of bleeding, both at the time of removal and, secondary, from a surface denuded of mucous membrane covered with granulations and crusts, as well as the ultimate formation of scar tissue with stenosis of the lumen of the trachea. With these facts in mind, the bronchoscopist may elect to remove the growth as completely as seems consistent with safety and with the avoidance of complications and treat recurrences as they may arise.

D'Aunay gives Lieutand credit for reporting the first tracheal growth in 1767, and states that Tuerch diagnosed such a tumor by mirror laryngoscopy in 1861. In prebronchoscopic days, most of the tumors reported were found on postmortem examination. The ratio of tracheal tumors to laryngeal tumors is reported as one to a hundred. Von Bruns found 147 tracheal growths reported in the literature up to 1898. In 1929, this number had reached 351 according to D'Aunay. With the more general use of the bronchoscope, the incidence of tracheal tumors reported is now relatively common, whereas formerly it was exceedingly rare.

The etiology of benign tumors of the trachea is unknown. It is possible that irritation by the pressure of stagnant

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purulent secretions may be a factor. As Jackson points out, the shape and size of the tumor may be affected mechanically by the expansion and contraction as well as the elongation and shortening of the tracheobronchial tree. The benign nature of these growths histologically does not alter the fact that they may give rise to severe secondary suppuration or even asphyxia.

The by-pass valve type of obstruction will give rise to wheezing, the check valve to emphysema, and the stop valve type to atelectasis. Of all the various types of tumors found in the air passages, the group of peculiar interest and controversy as to diagnosis and treatment is the group of adenomas or adenocarcinomas of low grade malignancy. The tumor reported falls in the class of adenomas.

The early symptoms of tracheal tumor are cough with slight mucoid expectoration and frequent attacks of respiratory infections. The onset is gradual and wheezing at the open mouth rather than through the chest wall is a characteristic sign. At times, hemoptysis is present but the sputum examination is usually negative for acid-fast bacilli. X-ray examination of the chest may be negative; however, if the tumor has encroached on the lumen of one of the main bronchi, atelectasis or emphysema may be present on the affected side. In this connection, the fluoroscopic and X-ray findings described by Manges may be noted in some cases, the ball valve effect of the tumor causing obstructive emphysema with increased illumination of the affected lung, depression of the diaphragm on the affected side, and a shifting of the heart and mediastinum to the uninvolved side on expiration and back to normal position on inspiration.

Successful removal of these tumors has been reported by Jackson by the use of the punch forceps and by Kernan by the use of surgical diathermy and the insertion of radon seeds. X-ray therapy is of value in the radio sensitive type. Care must be taken in the use of any method lest too much destruction of tissue lead to scar formation with resulting stenosis of the lumen of the trachea and possible occurrence of bronchiectasis or other pulmonary complications.

CASE HISTORY.

The patient was a 34-year-old white female housewife who was referred to the service of New York Hospital with a history of having been

treated for asthma for about two years. Five years previous to admission, she first noticed a slight hacking, nonproductive cough which persisted without relief. In November, 1938, the patient was examined and X-rays of the chest were taken but her symptoms were not fully explained. Two months later, in January, 1939, she first noticed dyspnea on exertion, orthopnea requiring two pillows for relief, and bronchial wheezing. These symptoms persisted with slight variations of severity. Some relief was experienced with adrenalin inhalations and injections. Allergic studies were made. The patient received 31 tests and seven inoculations and was found sensitive to chicken, beans, mackerel, feathers, goat hair, silk and sweet vernal grass. There was no seasonal change. Her cough remained essentially nonproductive, but sometimes a little phlegm was raised. No hemoptysis occurred. The patient became pregnant two and a half years ago, prior to admission, and during pregnancy her symptoms were intensified. The family history was noncontributory.

Physical examination on admission to the hospital revealed a well developed, well nourished 34-year-old female with slight evident respiratory distress and productive cough. Breathing appeared to be of the obstructive type with prolongation of both inspiratory and expiratory phases. The lungs were of normal resonance; breath sounds were diminished and prolonged, with occasional sibilant rhonchi. Wheezing was more noticeable on casual observation of the patient than through the stethoscope. Expansion of the chest was good and equal on both sides.

Fluoroscopic examination at this time revealed no evidence of obstruction by substernal thyroid or mediastinal mass. The lung fields were clear. Manges' sign was not present.

Laboratory findings were as follows: Repeated sputum examinations were negative. Blood and urine examination were negative. There was no eosinophilia. X-ray of the chest showed the heart, aorta and lungs to be normal.

The impression was that of an obstructive lesion of the trachea, and the patient was referred to the bronchoscopic clinic and examined on August, 1940. A 40x7 mm. bronchoscope was passed without difficulty, revealing a sessile mass, partially obstructing the lumen of the trachea about 22 cm. from the upper teeth, attached to the anterior and right lateral wall of the trachea. Sections of the tumor were removed with punch forceps. The patient was re-examined two weeks later under local anesthesia and diathermy was applied to the mass. Adequate breathing space was noted and the patient's symptoms improved until December, 1940, when, under avertin anesthesia, a 9 mm. bronchoscope was introduced and definite stenosis of the trachea, by a mass projecting into the lumen from the anterior and lateral wall 23 mm. from the upper teeth, was noted. A considerable portion of the mass was removed by punch forceps.

The pathological report was that of an exceedingly rare tumor composed of innumerable small masses of hydrochromatic opaque cells resembling chromaffin cells. "These masses are honeycombs with round cystic spaces, most of which appear entirely empty. In some areas, the tumor is more solid and shows a few cysts. There are occasional pseudoacini and the lumina contain homogeneous pale blue mucoid material. The tumor is covered by a well differentiated layer of respiratory epithelium. No mitotic figures are found and no malignant change is evident. This apparently represents a mixed adenoma of the trachea of the type which has been described by Hamperl and called by him oncocytoma."

In January, 1941, the base of the tumor was treated with electrocoagulation. The patient's airway improved, but some cough continued. Bronchoscopies were repeated in May and October, 1941, when more tissue was removed with punch forceps and diathermy. Following this, the patient had a period of comparative freedom from symptoms for about one year. She gained in weight. Her airway was adequate and she was

troubled only slightly by cough. In October, 1942, the patient again became dyspneic and under avertin anesthesia, a 9 mm. bronchoscope was introduced. Considerable encroachment on the lumen of the trachea by the tumor mass was noted. The tumor tissue appeared quite vascular and extended downward into the right main bronchus. This mass was again removed as completely as seemed practical with punch forceps and the base treated with electrocoagulation. During the past winter the patient has suffered from several attacks of bronchitis and rhinitis, but between these attacks her airway has been quite free.

Accordingly, between August, 1940, and October, 1942, six bronchoscopies were performed with removal of tissue by punch forceps, or by electrocoagulation, or by both methods. For a period of one year the patient experienced an interval of comparative freedom from symptoms. During this time no bronchoscopies were necessary. There has been no evidence of metastasis and the patient has had a normal confinement during the time of observation. The tumor was not radio sensitive.

In summary, a case of oncocytoma of the trachea in a patient treated for several years as an asthmatic is reported. Repeated bronchoscopies were performed with partial removal of the growth by means of punch forceps and surgical diathermy. There was temporary relief from symptoms and no evidence of metastasis. Bronchoscopy is indicated in patients having an "asthmatic wheeze" audible at the open mouth to eliminate the possibility of the presence of an intratracheal tumor causing symptoms resembling asthma.

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667 Madison Avenue.

DISCUSSION.

DR. NATHAN CHANDLER FOOT: I am very glad to discuss this case. The pathology of these tumors is a riddle, and because it is a riddle the theories as to their origin vary. As Dr. Palmer pointed out, the gross char-

acteristics of the tumor are very simple: It is a small growth which may be sessile, pedunculated, may grow in a recess in the bronchial tree, or may even be dumbbell shaped, growing in the tree and projecting into the lung. The results of the tumor are more important than the tumor itself on account of obstruction, ball valve action, etc. We have no clue as to where these tumors originate because they do not resemble the bronchial glands in any way, apparently representing a new type of cell. Hamperl has been studying tumors of this sort since 1930. He believes that in the salivary and mucous glands along the respiratory and alimentary tracts there are cells which increase in number with the age of the patient and gradually form small glandular complexes made up of rather opaque cells with pyknotic nuclei, and these cells, formerly known as pyknocytes, have been given the name of oncocytes by Hamperl because they are swollen. "Oncocyte" merely means a swollen cell. These cells tend to form acini with their surrounding ring of cells which form

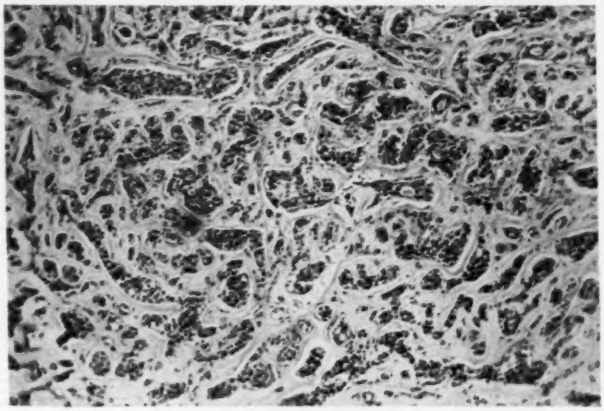

Fig. 1. Tracheal adenoma.

an almost regular circle. They are swollen, granular, have small pyknotic nuclei, and show granules in their cytoplasm which are sometimes capable of being impregnated with silver.

Some of these tumors grow in a very solid fashion, and then look so much like carcinoids that they are frequently called that. They are not identical with carcinoids of the alimentary tract, which do not exist in the respiratory tract as far as I know. In the case of this particular tumor, the early biopsy specimens which we obtained tended to grow in the carcinoid manner in solid masses, and in one case we were able to impregnate the granules in the cells so that the resemblance was striking. Biopsies taken at successive bronchoscopies showed the tumor to be taking on a more characteristic growth.

(Slide) Section of tumor showing masses. That was one of the early bronchoscopies. The tumor here has more resemblance to carcinoid than to oncocytoma, although there are cystic forms of gland-like growth which Hamperl has called "oncocytoma."

(Slide) The next slide is a carcinoid of the appendix, and I show it to give an idea of the resemblance between the two growths. The nuclei

of this particular tumor are larger and paler than those of the tracheal tumor.

There is only one other matter to discuss, and that is the question of whether these tumors undergo malignant change. I am pretty sure I have seen one case that became malignant. That was a case studied in

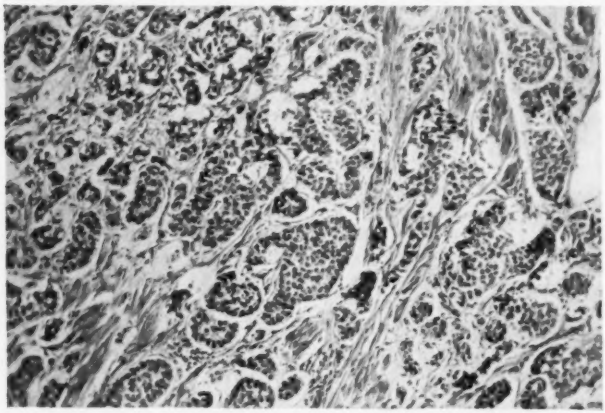

Fig 2. Carcinoid of appendix.

Boston, but these growths rarely undergo malignant change and bring about metastases. Many authorities feel that this does not happen at all. They believe that if the tumor becomes malignant the diagnosis was in error, even though it resembled this type of tumor.

SHRAPNEL IN SPHENOID SINUS CAUSING BLINDNESS. REMOVAL WITH RECOVERY.*

DR. IRVING B. GOLDMAN, New York.

Penetrating injuries of the nasal accessory sinuses by fragments of explosive shells present a special problem in war surgery. The present case is of interest since it illustrates some of the local and regional complications which may arise from the presence of shell fragments lodging in the sinuses. These complications may possibly be averted by early intranasal removal of the foreign body whenever feasible. In this instance severe visual loss which resulted was regained following removal of the shrapnel from the sphenoid and adjacent posterior ethmoidal cells into which it had become imbedded.

CASE REPORT.

The patient, a man 40 years of age, received a bomb injury 22 months before admission to the Semi-Private Pavilion of Mount Sinai Hospital. He had no recollection of events in the first week following injury, except that he was given fluids in his arms and legs. A shell fragment traveling in a horizontal and somewhat downward direction entered the right temporal fossa at a point opposite the zygomatic process of the frontal bone, traversed the right orbit just beneath its roof, proceeded through the ethmoid and its perpendicular plate and lodged in the anterolateral wall of the left sphenoid and posterior part of the ethmoidal capsule. Roentgenograms showed the metallic fragment to be 1½ cm. in diameter. On the right side, largely within the orbit, there were several irregular smaller fragments along the course of entry. Sectional radiography demonstrated the left fragment to be situated 6.5 cm. deep to the forehead (see Figs. 1, 2 and 3).

The patient complained of total loss of vision in his right eye and marked diminution of vision in the left. After spending some time at hospitals and convalescent homes in London, Liverpool and Scotland, he again went aboard a boat to carry on despite his visual handicap.

Four months previous to admission to the hospital he began to experience further loss of vision in his left eye. This progressed to such an alarming degree that a physician who examined him in Jamaica, British West Indies, on Aug. 22, 1942, had him sent to New York for consideration of operative removal of the foreign body.

Examination showed an emotional individual who was generally well except for a polyneuritis of his lower extremities, possibly on a basis of avitaminosis. Ophthalmological examination showed total blindness and

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Fig. 1. Anteroposterior view showing metallic fragments and deformity of greater wing of right sphenoid resulting from fracture by shrapnel.

Fig. 2. Laminogram demonstrating left fragment to be 6.8 cm. deep to the forehead.

absence of light perception in the right eye. The vision in the left eye was reduced to counting of fingers at one foot, corrected with +1.50 sphere to 20/70. The extraocular movements in this eye were normal.

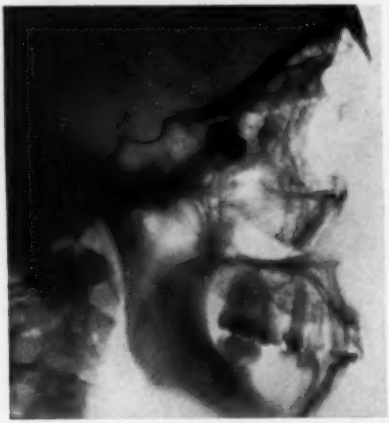

Fig. 3. Lateral view before removal of shrapnel.

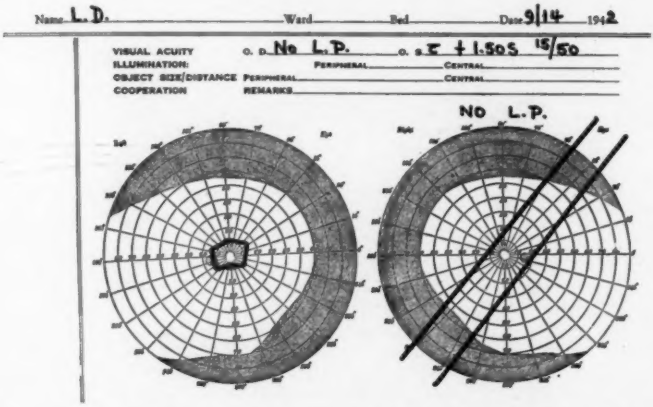

Fig. 4. Visual fields before operation showing left tubular and right absent field.

The right eye could not be moved beyond the midline on looking to the right, nor could it be depressed. There was no difficulty in the upper gaze. External examination was negative except for tenderness over the left eye. The right pupil was in mid-dilatation, irregular, did not react to light, but reacted slightly consensually and during convergence, though not as promptly as the left eye. The left pupil was normal in size, reacted to light, but reacted only slightly consensually. Fundus examination showed the right disc to be pale and grayish, with increase in the cup-

ping and clear margins. The left disc appeared normal. Both maculae showed pigmented and atrophic changes. Below and temporal to the discs there was a linear area of chorioretinitis. The retinal vessels were normal in both eyes. The field of vision of the left eye was constricted

Fig. 5. Anteroposterior view after removal of foreign body showing clouding of sinuses.

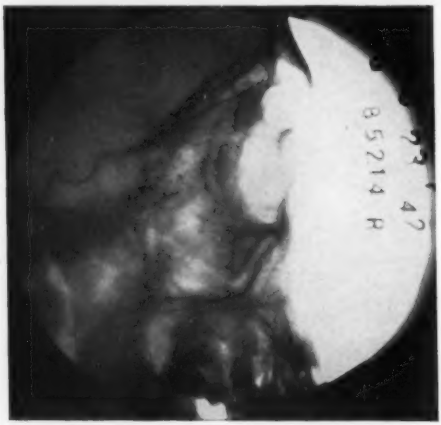

Fig. 6. Lateral view after removal of shrapnel.

to within 20° of the point of fixation and was of the tubular type. The color of a 3 mm. object could not be recognized but all of the 10 mm. color test objects were easily discernible. The corrected vision in the left eye was 20/70 (see Fig. 4).

The diagnosis was: *1.* paralysis of the right external and inferior rectus muscle, most probably due to intraorbital trauma; *2.* right pupillary changes due to loss of vision; *3.* right primary optic atrophy; *4.* bilateral macular retinal degeneration of the type seen in head trauma; *5.* visual changes in the left eye due to macular degeneration with the possibility of the presence of hysteria.

An intranasal operation was performed under local anesthesia. The left middle turbinate was removed. At a distance of 6.8 cm. from the orifice of the nares a grayish metallic mass was seen jammed in the lat-

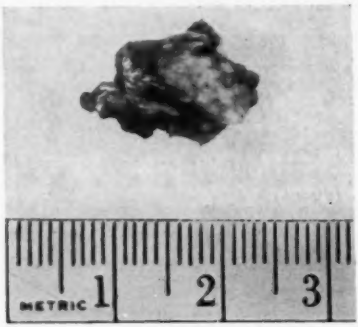

Fig. 7. Photograph of shrapnel after removal.

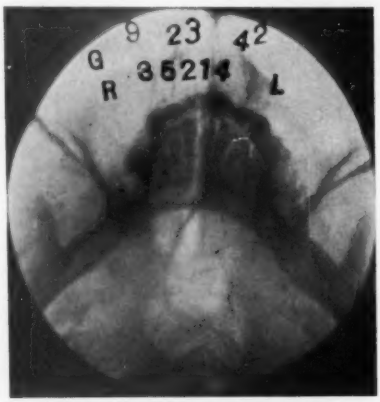

Fig. 8. Submentovertex position showing large left sphenoid extending to the right side.

eral wall of the sphenoid. It occupied the left sphenoid sinus anteriorly and extended somewhat into the posterior ethmoidal region. The anterior wall of the sphenoid was gone and there was a fair sized perforation of the perpendicular plate of the ethmoid and rostrum of the sphenoid. Gentle efforts to release the firmly imbedded shrapnel by means of fine hooks and forceps were of no avail. In order to get a better purchase on the foreign body, a few neighboring cells were removed. The metallic

mass was then grasped with a heavier forceps and by means of a rocking motion was unlocked from its bed. Thin pieces of bony septa were attached to the shrapnel. The left sphenoethmoidectomy was then completed. In a small posterior ethmoidal cell, adjacent to the region of the foreign body, pus was found which on culture grew staphylococcus aureus A (see Figs. 5, 6, 7 and 8).

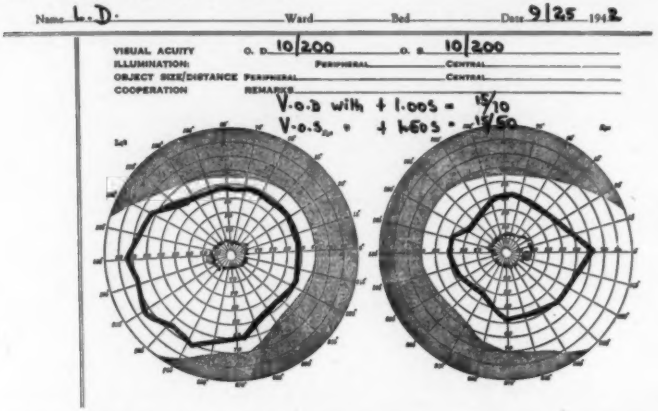

Fig. 9. Visual fields two weeks postoperatively showing right field greatly improved, left field practically normal.

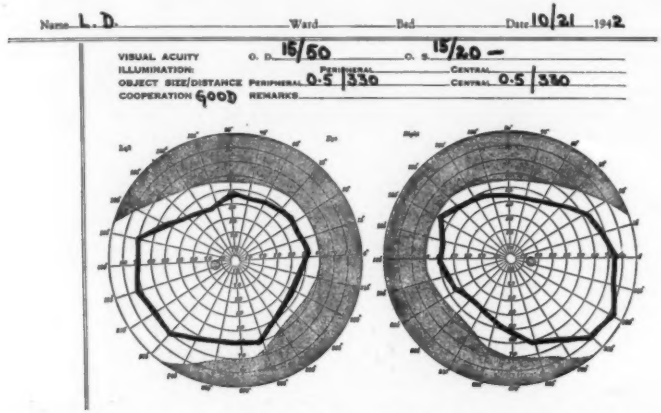

Fig. 10. Visual fields six weeks postoperatively showing both fields virtually normal.

Immediately following removal of the foreign body, and while the patient was still on the operating table, he exclaimed that he could see. Finger-counting tests confirmed this. Repeated ophthalmological examinations two weeks later showed the left visual field to be within normal limits, and return of the color fields. There was considerable increase in the field of vision in the right eye, and the field for form had returned

so that its peripheral limits were now within 50° to 60° of the central point of fixation. Color vision was normal in both eyes. Vision in the right eye was 10/200; with correction was 20/100. Vision in the left eye was 10/200; with correction it was 20/70 (see Fig. 9). Visual fields taken a month later showed both to be within normal limits. Vision with correction in the right eye was 15/50; vision with correction in the left eye was 15/20 (see Fig. 10).

COMMENT AND CONCLUSION.

The prompt restoration of vision following extraction of the shrapnel suggested the possibility that one was dealing with hysterical amblyopia. This idea was augmented by the tubular vision in the left eye, which ophthalmologists generally presume to be of hysterical origin. As a matter of fact, the centrally contracted visual field in the left eye was by itself at first considered to be a contraindication to operation because of the chance that the entire clinical picture was functional. On the other hand, prompt return of vision after operation is sometimes seen in other conditions such as retrobulbar neuritis, and optic neuritis following sinus operations when infected foci in the sinuses are responsible for their presence. Great importance must be placed on the position of the optic nerve in its relation to the sphenoid sinus, in the evaluation of the contralateral as well as unilateral symptoms. Not only may the optic nerve lie against the lateral wall of the sphenoid or bulge into it, it may even lie free in the sinus. Removal of pressure of the shrapnel on the lateral wall, especially where there has been loss of wall substance due to pressure atrophy, particularly in the presence of a projecting nerve, would explain the rapid return of vision. A large sphenoid, such as this left sphenoid, may swing around the smaller right sinus to be in close proximity to both optic nerves. The recovery of vision in the right eye as well as the left could then be explained by the size and position of the sphenoid sinus; that is, on an anatomical rather than on a functional basis.

121 East 6th Street.

DISCUSSION.

DR. KNUT HOEGH HOUCK (by invitation): It has been my good fortune to work with Dr. Goldman on the case he has just presented, which, to our knowledge, is the first one of its kind to be reported in this country. As Dr. Goldman has described, there were several unusual findings and the operative procedure was not undertaken without considerable discussion. It was Dr. Goldman's wish that I take up in discussion the neuro-

psychiatric details, the most important of which he has discussed; namely, the visual symptoms and the pupillary reflexes.

Although the patient had not been in military service, the problem he presented is one strictly of wartime. This man was a ship's cook on a Dutch merchant vessel which was bombed as it lay at anchor off the Island of Jersey in November, 1940. His regular occupation was that of a diamond polisher in Belgium, but since 1926 when things became dull in the diamond trade, he had gone to sea as a ship's cook. Although he had no memory of events of the week subsequent to his injury, he later learned that his captain had taken him in a small boat to the English mainland, where he was treated in a London hospital for about two months. He knows that he was given either transfusions or infusions and was told that operative procedure was not undertaken because at the time London was being bombed severely. He recovered sufficiently to go to a hospital in the interior of England for about four months, after which he was allowed to go to his home in Scotland for a period of six months. All this time the vision in the right eye appeared to be completely absent and that in the left eye was very defective. He also suffered pain in the left eye and forehead. His strength and vision improved so that in October, 1941, he was sent to Liverpool to join his ship; however, he was kept there inactive until June, 1942, when he finally joined a ship which brought him in convoy to America.

It was while on this ship that the vision in his left eye became markedly impaired and the pain in that area more severe. When the ship finally docked in Jamaica the physician there took X-rays and discovered the optic atrophy in the right eye, and it appeared that he was developing a retrobulbar neuritis in the left eye. The patient was consequently put on a plane and sent to New York for immediate surgical care. Dr. Sigmund Falk, who was in charge of medical affairs for the shipping company, was given to understand by telegraph and telephone that the foreign body lay within the cranial cavity. The neurological survey was, therefore, a first consideration; but the examination revealed no evidence of cranial injury and the X-ray established the location of the metallic fragment in the left nasal passage impinging upon the left orbit.

The only neurological finding not mentioned by Dr. Goldman was a subjective impairment of superficial sensation throughout the right side of the body. The patient is rather insignificant in appearance. He was most co-operative, very polite and gave the impression that he was conducting himself as a courageous war casualty; however, he did give way to tears on one or two occasions and his wincing when the left eye was palpated or even when Dr. Goldman sprayed cocaine in the left nostril prior to examination gave one the definite impression that the patient was somewhat oversensitive and that at least unconsciously there was a good deal of apprehension. He fitted quite well into the rôle of the modest hero and was so accepted by the nurses, by a young daughter of the patient in the next bed and also, I believe, by his physicians.

It would have been difficult to obtain a more complete psychiatric history because of considerable language difficulty. Laboratory tests showed a negative blood Wassermann, normal urinalysis, 75 per cent hemoglobin and 3,850,000 red blood cell count. In evaluating the eye findings, it was my impression that there was potential vision in the totally blind right eye, because when light was thrown into that eye there was a good consensual reflex of the left pupil, which meant to me that the fibres carrying light sensation must have been intact through the right optic nerve to the connections in the superior colliculi in the brain stem; and as far as I could find there was no evidence of any lesion along the optic tracts behind where those fibres left it, which could explain the monocular blindness and the tubular vision in the left eye. In other words, there was no evidence of destructive lesions in either temporal or occipital lobes. I

felt, therefore, that if the blindness was of a hysterical nature the operative procedure would at least be of sufficient magnitude to play a major rôle in suggestive therapy. I was not thoroughly convinced of this premise, but it would seem most logical that if the fragment was causing direct or indirect pressure upon either optic nerve, removal of the fragment itself was practically imperative, especially since it could be reached with little danger of penetrating the orbit or cranial cavity. In order to lay a certain groundwork, through suggestion, for return of vision in the event the blindness was functional, the patient was told it was most probable that he would again be able to support his wife in Scotland, return to her a cured man, and even take up his trade in the diamond industry again after the war was over.

It was, therefore, most gratifying to note that while the patient was still in the operating chair he regained the ability to count fingers at 10 feet and that gross testing of the visual fields in both eyes showed them to be within normal limits. When this was demonstrated to the patient, he broke into heavy sobbing, as though the strain of the previous two years had been finally resolved.

After about eight weeks' convalescence, the patient again returned to duty as a ship's cook, having made two consecutive trips on troop ships to North Africa, where he and another cook were responsible for feeding 2,000 enlisted men three meals daily, which meant working often from 3 A.M. until 11 P.M.

I realize that this case is only a single one, but it is significant not only because it is the first of its kind to be reported in this war but also because, I think, it demonstrated pretty clearly that the tolerant attitude of everybody concerned in the case was decidedly helpful in arriving at the most desirable mode of management and in the favorable outcome. The procedure undertaken was perhaps not ultrascientific in that one method was chosen to the exclusion of the others, but each of us gave the others the benefit of the doubt, whether it was Dr. Falk, who referred the case to us, Dr. Last, who did the ophthalmological investigation, Dr. Goldman or myself.

In summary, I may say that this unusual case of Dr. Goldman's of a 40-year-old Belgian ship's cook who was wounded in November, 1940, and operated upon in September, 1942, presents a most gratifying surgical and psychiatric result and is interesting because of its timeliness in the present war situation as well as because of its dramatic personal appeal.

OTOGENOUS ENCEPHALITIS.*

DR. CLARENCE H. SMITH, New York.

Various observers have suggested the possibility of nonsuppurative encephalitis being a complication of otogenous infections. This condition is advanced as a distinct entity.

The clinical picture is one of brain abscess with localizing signs, but in which brain puncture is fruitless. In spite of the serious signs and symptoms and the lack of success on exploration, the tendency is toward complete recovery.

Otogenous encephalitis is the result of an infected focus in the temporal bone or in the nasal sinuses.

Borries,[1] in 1921, was the first to consider that otogenous encephalitis exists independently, differing from the encephalitis which forms the first stage of a brain abscess, claiming that the processes are pathologically different.

Borries[1] subdivides the inflammations of the brain into simple encephalitis, hemorrhagic encephalitis and purulent encephalitis. The simple encephalitis is the most benign form. Hemorrhagic encephalitis frequently occurs as an autonomous disease without tendency to suppuration. Purulent encephalitis means a brain abscess, and it sometimes develops from hemorrhagic encephalitis.

Oppenheim,[2] in 1900, and Voss,[3] in 1902, reported cases of nonpurulent otogenous encephalitis complicating ear inflammations. They considered the condition as a developing stage in a brain abscess.

Adson,[4] in 1924, described similar cases. He thought the condition was localized encephalitis, and named it "pseudo-brain abscess."

Yerger,[5] in 1925, and Symonds,[6] in 1927, described cases which showed increased intracranial pressure and focal brain disease complicating aural infections, without meningitis and without brain abscess, as evidenced by exploration, the cases finally recovering. Symonds,[6] too, suggested that the condi-

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tion of localized nonsuppurative encephalitis may be a stage of inflammation which is arrested and resolves.

Cairns,[7] Key-Alberg,[8] Voss,[9] Colledge[10] and others have reported similar cases. Eagleton[11] thinks that otogenous encephalitis is probably more frequent than suspected.

In a study of 326 cases with neurological complications arising from infections of the temporal bone and paranasal sinuses, Yaskin[12] found four which he considered as having localized nonsuppurative encephalitis.

The pathology of this condition is necessarily rather meager since these cases generally recover.

Borries[1] says that in otogenous encephalitis there are present microscopically detectable perivascular leucocyte infiltrations. There are visible hemorrhagic infiltrations of the cerebral tissue in hemorrhagic encephalitis; this variety is more serious than the simple encephalitis. The location of these inflammations is generally in the temporal lobe; it is far less frequent in the cerebellum.

Jerlang[13] says that in many cases where the pathological process are located in the silent areas of the brain, or perhaps are very limited in extent, the symptoms are so vague and atypical that the affection is either not recognized at all or is misinterpreted.

The onset of otogenous encephalitis may be slow or sudden. Children and young adults are most often affected. It is seen in acute and chronic otitis.

Diagnosis: The diagnosis of otogenous encephalitis is always difficult. Borries[1] says that this disease is characterized by "abscess symptoms without abscess."

Focal brain signs are the most constant accompaniment. Yaskin[12] enumerates these as hemiplegia, central facial palsy, Jacksonian convulsions, aphasia, sensory symptoms, hemianopic defects and cerebellar signs. The fact that these signs are also to be expected in brain abscess and extradural abscess complicates the situation.

There is nothing characteristic about the spinal fluid findings. There may be no cells or there may be a turbid fluid with many cells. There may or may not be an increase in pressure. The findings on lumbar puncture give little help in the differential diagnosis.

Oppenheim,[12] in 1897, stressed the point that high temperature in otogenous encephalitis is to be expected. This is in contrast to the moderate temperature seen in brain abscess.

Jerlang[13] points out that the pulse rate is generally proportionate to the temperature and that true bradycardia has not been observed. Headache is less marked in this affection than in cerebral abscess.

Otogenous encephalitis is often mistaken for a brain abscess and the correct diagnosis is made only after unsuccessful exploration of the brain in the presence of outstanding focal symptoms, with the subsequent complete recovery of the patient. An electroencephalogram, if obtainable, may be of service in suggesting a brain abscess, and it could be used as a diagnostic aid. If the electroencephalogram produces an abnormal pattern, limited to one region of the brain, it would be a factor in diagnosing the condition as a brain abscess. The reaction in encephalitis is more diffuse.

Sometimes the symptoms may be caused by the presence of an epidemic or influenzal encephalitis in a patient suffering from ear disease. The knowledge of epidemic encephalitis being present in the locality at the time, and the presence of a virus in the spinal fluid in epidemic encephalitis is helpful in the diagnosis. In epidemic encephalitis, the brunt of the pathological changes generally falls on the brain stem and basal ganglion, although any part of the brain may be involved.

The treatment of otogenous encephalitis consists in the early and thorough eradication of the infective focus.

In cases of apparent brain abscess there is danger in excessive needling of the brain in an effort to locate the pus. Jerlang[13] says that puncture of the brain does not have an unfavorable effect on the encephalitis. Eagleton,[11] in discussing this, says: "Anyone who has microscopically examined the cerebral tissue along the track of a diagnostic puncture must appreciate the falsity of such a statement. Any puncture is attended with some danger, especially in the presence of a suppurative process in the neighborhood."

The prognosis in autogenous encephalitis is good, not only as to life but also with regard to the intracranial symptoms which generally clear up entirely.

A recent case may be of interest, as it illustrates the disease referred to with the attendant problems of diagnosis and treatment.

Carol A., female, age 11 years, was first seen on admission to the Bronx Eye and Ear Infirmary on Aug. 19, 1942.

Twenty-three days previously, after earache following swimming, her ears began to discharge pus. This discharge ceased seven days before admission. A sulfonamide had been given to her from the beginning of the ear symptoms. She had complained of earache for several days. Three days before admission, the patient had lapsed into a deeply stuporous state and had a convulsion, which, from the parents' description, was general. Later that day she had convulsive seizures of the face on the left side, and in the left arm. During the three days mentioned, she had several left-sided Jacksonian convulsions. Vomiting was present.

Examination revealed a well nourished child, in a semicomatose state. The skin was dry and hot, evidently due to lack of fluids. She maintained a left-sided cerebellar attitude to which she always reverted after her position was changed.

Examination of the ears showed an absence of discharge, reddened, thickened drums, and no mastoid tenderness. Blood chemistry examination revealed a slight elevation of urea-nitrogen and creatinine. This could be accounted for by the absence of fluid intake during the preceding few days. A small number of red blood cells and a trace of albumin were found in the urine.

Spinal fluid examination showed a clear and colorless fluid with four cells per cmm., globulin and sugar in normal amounts. The pressure was normal.

The temperature on admission was 101.6° and was consistently low during her illness. The pulse rate followed the temperature curve; bradycardia was not present at any time.

The neurologist found there were no signs of meningeal irritation. Paresis of the left facial nerve was present. Examination of the left upper extremity revealed weakness. Bilateral Babinski reactions were present. All deep reflexes and abdominal reflexes were absent. The fundic discs were seen and revealed bilateral blurring on the nasal side.

During the examination a Jacksonian convulsion occurred. This consisted of clonic movements of the left arm, then the face on the left side, and lastly the left leg.

The neurologist's impression was that she possibly had a right temporosphenoidal abscess, causing upward pressure on the anterior central gyrus, irritating the face and arm centers. He advised observation to make sure that the condition was not caused by encephalitis, which had not matured to abscess formation. He thought the findings in the blood and urine were due to chemotherapy and to bladder neglect during the days of coma.

The Roentgenologist reported, "On the left side there is a normal mastoid, with slight decalcification of the septa and moderate cloudiness over the entire process. On the right side there is considerable cloudiness over the entire process. The cells in the upper part of the mastoid are completely destroyed and the intercellular septa near the tip show a moderate decalcification."

On the day after admission, the patient had a simple bilateral mastoidectomy. Considerable necrosis throughout the mastoids was found, including the inner table over the sigmoid sinuses.

Parenteral fluids were given in intravenous injection of 5 per cent glucose in saline solution, and daily lumbar puncture was instituted.

The patient's condition slowly improved. Two days after the operation, she gradually came out of her coma, recognized her mother and

seemed to orient herself. On the third postoperative day, speech returned and she asked for food. From that day her progress was rapid and she was able to leave the hospital in excellent physical and mental condition on the ninth postoperative day.

Comment: This patient presented the symptoms of a cerebral abscess and the focal signs of a temporosphenoidal abscess. These symptoms and signs gradually disappeared when the infective focus in her mastoids was removed. This case cannot be explained unless the possibility of nonpurulent otogenous encephalitis is taken into account.

In a case with general and localizing signs of cerebral abscess, a negative exploration need not necessarily mean a missed abscess. The picture might very well be caused by the condition we are considering. The knowledge that there is such a possibility should prevent the surgeon from too many explorations in various directions, in his desire to find an abscess.

Exploration of the brain should be delayed as long as apparent safety permits. This will promote encapsulation if an abscess exists. If only simple nonsuppurative encephalitis is present, removal of the infective focus may be all that is necessary to promote the recovery of the patient.

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140 East 54th Street.

LARYNGECTOMY FOR SOME EXTRINSIC CANCERS OF THE LARYNX.*

DR. MERVIN C. MYERSON, New York.

Extrinsic cancers of the larynx are encountered much more frequently than the early intrinsic forms in a large hospital service. It has always seemed to the writer that an attempt should be made to rescue at least some of these cases. In recent years it has been our policy to operate upon cases in which it was felt that there was even a slight chance of success. Have we the right to ask whether we are not too conservative? Are we not still following indications set many years ago when the mortality of laryngectomy was high and the technique was not yet perfected?

Many patients who receive radiation therapy develop perichondritis and necrosis of the laryngeal cartilages. Although the postoperative wounds of such patients are slow to heal, it is felt that previous X-ray therapy should not be a contraindication to surgery. Only if the skin and subcutaneous tissues have been so badly devitalized that there is no longer any chance of healing should such cases be eliminated.

The histories of two cases are cited to illustrate the value of surgery in the apparently hopeless case.

Case 1: H. K., a man aged 65 years, was admitted to the hospital complaining of hoarseness which had lasted for two years. Physical and X-ray examination seven months before admission suggested the possibility of pulmonary tuberculosis and at that time he was then admitted to a tuberculous hospital. His larynx was considered tuberculous. Because his sputum was negative and continued to be so, he was referred to the hospital. He had lost much weight.

Examination at the time of admission disclosed the presence of healed pulmonary tuberculosis. Except for his larynx the patient appeared to be in very good health. Laryngeal examination revealed a greatly constricted airway which was causing some degree of dyspnea. The epiglottis had been replaced by a large fungating mass which occupied not only the introitus of the larynx but also a part of the base of the tongue. The mass was large, encroaching upon the interior of the larynx. The structures below the level of the cancer could not be visualized. Biopsy was performed and, because of increasing dyspnea, tracheotomy was done. The laboratory reported a well differentiated squamous cell carcinoma.

Despite the extent of the cancer, a radical operation was contemplated which would include removal of the larynx and resection of the involved base of the tongue. Accordingly, a laryngectomy was performed.

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In order to provide better access to the tongue base, the hyoid bone was resected. A portion of the tongue base was removed. The neoplasm was about two inches in diameter, extended down the left side into the subglottic region and weighed more than the larynx itself.

The patient enjoyed a very mild postoperative course. He was out of bed on the second day and completely well at the end of three weeks. More than a year has elapsed since the operation and he enjoys excellent health.

Case 2: A man 54 years of age was referred to the hospital because he had been hoarse for one year. He was otherwise in excellent health. In addition to a cancer of the larynx, which occupied the anterior two-thirds of the supraglottic area on each side, there was a mass of involved lymph glands in the left jugular node area. Biopsy revealed the presence of an immature squamous cell carcinoma.

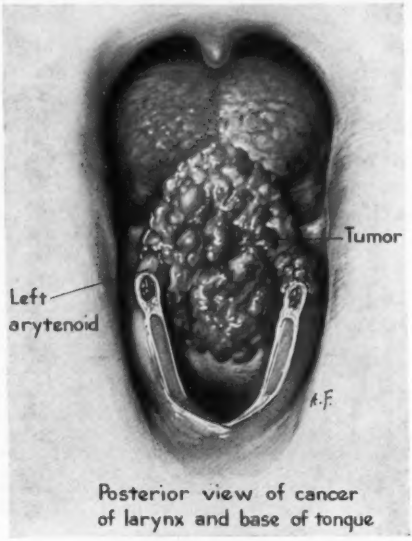

Fig. 1. Case 1.

Radical resection of the involved glands was performed. This was followed one week later by extirpation of the larynx. Because of the location of the cancer, the cricoid cartilage was not removed.

One year after operation, an enlarged lymph node appeared alongside the tracheal stoma. This disappeared after X-ray therapy. The patient enjoyed excellent health for three and one-half years after the laryngectomy, when he developed a metastatic lesion in the chest, which was the cause of his death.

Hemilaryngectomies and laryngectomies have been performed upon necks which have been exposed to heavy X-ray dosage. We have come to the conclusion that it is best to avoid operation upon tissues which are stiff, telangiectatic

and devitalized. Where such conditions do not exist, however, there is reason to expect healing, though it might be delayed.

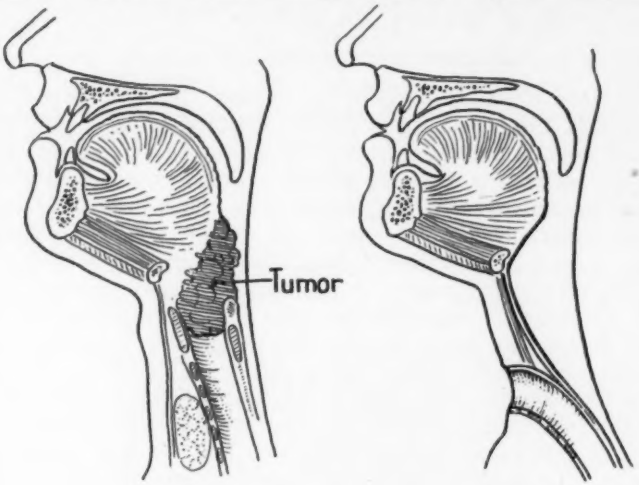

Fig. 2.

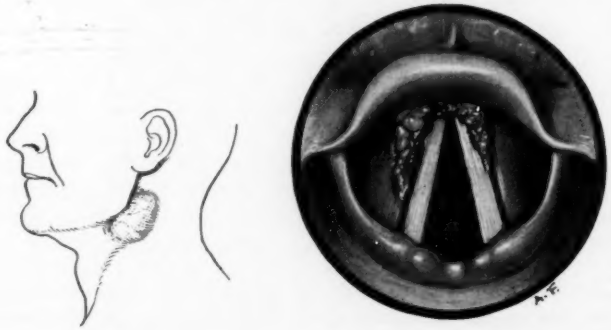

Fig. 3. Case 2.

This presentation is a plea for surgery in certain cases which have heretofore been considered inoperable.

136 East 64th Street.

THE AUDITORY OR EUSTACHIAN TUBE.*†

DOROTHY WOLFF, Ph.D., New York.

At the close of the last war it was fully recognized by aviators that difficulty with the Eustachian tube and the maintenance of balanced air pressure on the two sides of the drum membrane were problems of importance. Following that war, Rich, at the suggestion of Dr. Howell, of the Johns Hopkins Medical School, performed a series of investigations on the Eustachian tubes of dogs. To date these observations, made in 1920, appear to be the most accurate statements in the literature in regard to the gross anatomy and function of the Eustachian tube. Rich concluded that: *1.* normally the tubes are closed; *2.* the tensor veli palatini, innervated by the Vth nerve, is the only functional muscle of the tube; *3.* the tubes are opened during the swallowing, yawning and sneezing reflexes, and are best ventilated by the yawning reflex. Armstrong, Holmgren, Van Dishoek, Campbell and Hargreaves have all made important recent clinical observations in regard to the function of the tube in man.

Microscopists have somewhat lagged behind these investigators in their contributions to this important subject. This fact leads again to a realization of the necessity of routine collection of postmortem specimens for the proper examination of all regions associated with the petrosae. Lierle, Guild, Jones, Hemsath, Polvogt, Babb and Szaz, *et al.*, have contributed to this field. Up to the present time, however, no microscopic sections have been presented in the literature, illustrating the interesting clinical observations recorded by Shambaugh, Pearlman, *et al.*, of persistently patent Eustachian tubes, or marked atrophy of tubal tissue.

The obscure location, diagonal course, and angular descent of the tube render dissection and demonstration of this important structure a difficult matter. Microscopic serial sections of the tubal region, taken throughout the life span from

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*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, March 18, 1943.

†Data for the observation in this paper were collected in the Oscar Johnson Institute, St. Louis, and in the Carnegie Institution for Embryology, Baltimore.

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embryo to adult, give an accuracy of conception of the anatomy otherwise unobtainable. The accumulation of well catalogued pathological sections of this region accompanied by detailed clinical histories with statements in regard to the presence or absence of such seemingly unimportant symptoms as tinnitus, bruit and deafness, taken from the wards of a large general hospital, should eventually yield valuable statistical data.

As has long been known, the Eustachian tube develops embryologically from the first gill pouch and is, therefore, a diverticulum of the pharynx. Its definitive form is well established in the second embryonic month as a rather deep and extensive groove on the lateral wall of the pharynx. In cross-

Fig. 1. The Eustachian tubes leading from the pharynx to each middle ear which is filled with mesenchyme at this stage of development. Note the three ossicles. Carnegie collection, No. 7864, 8 weeks, 2 days.

section the two tubes appear as blunt, slightly curved diverticulae, extending superolaterally from the pharynx. The first figure is from a specimen in the Carnegie Collection showing the tube as it appears at the close of the second embryonic month. Here we can recognize the rudiments of each of the three ossicles as well as the invagination of the external auditory canal. In a two and one-half months embryo the tube presents a slight narrowing midway in its course already indicating the future isthmus. At this early period there is a slight differentiation of epithelium of the tube but no ciliated cells are as yet discernible, although these can be seen in parts of the nasal region. It is important to

note that at no time in its development is there mesenchyme in the lumen of the tube.

In the latter part of the third embryonic month the muscles in anatomic relation to the Eustachian tube are clearly established. Fibres of the *tensor veli palatine* may be seen passing from the lateral side of the lumen, around the hamulus of the pterygoid process of the sphenoid to meet the mate from the opposite side in the region of the soft palate. The fibres of the *levator veli palatini* pass from beneath the tube directly upon the level of the soft palate, likewise to meet those of the opposite side.

The fourth fetal month marks the period of greatest development in the auditory tube. During this month the cartilaginous support is first developed. This arises from four centers of chondrification which fuse to form the medial cartilaginous wall. No superior cartilage exists at this time. Ciliated columnar epithelium first appears lining the lumen of the tube in this month. Goblet cells also are present. A tortion of the tube occurs at this age causing a change in the direction of the long axis of the tube. The long axis is in vertical plane down to the narrowed section (isthmus). Below this level it assumes a horizontal plane. This condition is not maintained throughout later development.

In the fifth fetal month glands (mixed in type) are seen along the medial wall of the Eustachian tube toward the pharyngeal lumen. Rugae begin to appear. The extension of the lumen of the tube has advanced toward the middle ear so that pneumatization of that structure has proceeded posteriorly to the region of the malleus.

In the sixth fetal month the tubal cartilage extends toward the tympanum even beyond the apex of the cochlea. This condition is maintained at birth and was observed in an infant as old as 10 days.

The bony portion of the Eustachian tube was first observed to be well developed in the third month of infancy. As the petrous apex develops and elongates, it evidently pushes the cartilaginous portion of the tube downwards in front of the elongating bony tube. Never does tubal cartilage show any tendency to ossify and thus take part in the development of the bony portion of the tube. In old age, however, ossification

of the cartilage of the tube at the isthmus is sometimes observed.

In the six-months infant, differentiation of the epithelial lining of the tube is clearly established. The inferior portion or gutter of the tube bears tall, ciliated columnar epithelium. Along the side walls of the tube the cells are reduced in height somewhat and over the roof they are almost cuboidal. As the tube enters the middle ear the cells all become cuboidal and nonciliated.

For ideal studies of the Eustachian tube it is advantageous to remove only one petrosa, severing the sphenoid block in the midline in order to procure as complete an extent of the

TABLE I. EUSTACHIAN TUBE AT ISTHMUS.

	Race	Sex	Ear	Age	Height	Width
1.	N	M	Rt.	2 days	1.75 mm.	.75 mm.
2.	W	M	Lt.	5 mos.	2.5 mm.	1. mm.
3.	W	F	Lt.	6 mos.	2. mm.	.32 mm.
3.	W	F	Rt.	6 mos.	2.4 mm.	.3 mm.
4.	W	F	Rt.	8 mos.	2.4 mm.	1.5 mm.
5.	W	M	Lt.	4½ yrs.	3.5 mm.	.4 mm.
6.	W	M	Lt.	5 yrs., 8 mos.	2.2 mm.	.75 mm.
6.	W	M	Rt.	5 yrs., 8 mos.	2.4 mm.	2. mm.
7.	W	M	Lt.	12 yrs.	1.9 mm.	.2 mm.
7.	W	M	Rt.	12 yrs.	1.6 mm.	.4 mm.
8.	W	F	Rt.	28 yrs.	5. mm.	2.5 mm.
9.	W	M	Lt.	31 yrs.	4. mm.	3.4 mm.
10.	W	F	Lt.	42 yrs.	4. mm.	2. mm.
11.	W	M	Lt.	44 yrs.	5. mm.	1.-1.5 mm.
12.	N	M	Rt.	45 yrs.	3. mm.	.8 mm.
13.	W	M	Rt.	Adult	4.8 mm.	.8 mm.
14.	W	F	Rt.	111 yrs.	3.9 mm.	2. mm.
14.	W	F	Lt.	111 yrs.	5. mm.	1. mm.

cartilaginous tube as possible. Vertical sections cut at right angles to the long axis of the petrosa offer the best material for comparative studies of the relative size of the tubal lumen at the isthmus in various ages. In order to establish a norm for a single age group, measurements should be made on large numbers of specimens for each decade. Accumulations of more material by many investigators will be necessary before any accurate conclusions can be drawn. Table I gives measurements of the tubal lumen at its narrowest portion in a series of 14 tubes, from individuals ranging in age from two days to 111 years (authenticated age). Fig. 2 illustrates a series of projection drawings of the isthmus of the tubes from cases ranging in age from stillborn (sixth fetal month)

Fig. 2. The isthmus of the Eustachian tube from fetus to old age.

to old age. It will be seen from these observations that the infant tube is no larger and wider than the adult tube. Lack of development of bone surrounding the tube at this early age, bone which might offer some resistance to the passage of a bougie, has evidently led to the erroneous conclusion that the lumen of the infant tube is larger than that of the adult. Frazier has warned against the hazards of bouginage.

In regard to the presence or absence of lymphoid tissue within the Eustachian tube, it must be stated that in a study of 250 pairs of temporal bones, an organized lymph nodule

Fig. 3. Eustachian tube at the isthmus, showing an exceptionally thin tegmen over the tube. Obviously this has occurred as the result of extensive pneumatization, a peritubal cell having developed in this location. The condition was bilateral. The patient died of meningitis of otitic origin on the right side. The route of extension was not at this point, however. W.U. 7354 Lft. 860.

(Gerlach's tubal tonsil) was found in only one specimen and in only one tube of that case (a six months infant). Forty-seven petrosae were removed at autopsy with observations on the tube in mind so that particularly long blocks (anteriorly) were procured. In none of these was a tubal tonsil observed, although the specimens covered a wide age range.

Numerous organized typical lymphoid nodules are routinely seen in Rosenmüller's fossa just posterior to the torus tubarii. In no microscopic sections were these observed to effect a tubal closure.

Great anatomic variation is exhibited in the pneumatization of the peritubal region and in the proximity of the tube to the internal carotid artery, as has been previously demonstrated by other investigators. Fig. 3 illustrates an anomalous condition in which the tubal lumen at the isthmus extends superiorly to a very thin tegmen. The cartilage normally placed above the tube is displaced laterally.

As is to be expected, the mucosal lining of the tube always shows pathological change in the presence of an otitis media. It is not generally realized, however, that frequently the portion of the tube below the isthmus will show direct evidence

Fig. 4. Stratified squamous epithelium in the Eustachian tube. This was a bilateral condition, occurring in an infant with cleft palate.

of reparative processes (in response to pharyngeal medication presumably), while the portion above the isthmus may exhibit a devastating pathological condition.

Direct pathological extension from an infected tube into the marrow of the petrous apex may produce an osteomyelitis of that region; or it may lead into pneumatic cells of the apex with the development of an extensive accumulation of pus in these cells and typical apicitis. Again, extension may be toward the carotid canal forming an abscess in the carotid sheath, which in turn may erode the internal carotid artery.

In the examination of 250 pairs of temporal bones only four tubes were found in which stenosis of the Eustachian tube could be demonstrated. Unfortunately none of these had a clinical history giving any statement in regard to hearing or tinnitus. Excessive retraction of the drum membranes was not evident in the microscopic sections.

Fig. 4 illustrates a tube from an infant in which isolated patches of stratified squamous epithelium occurred bilaterally in the bony Eustachian tube. Of added interest is the fact that this occurred in a case with a history of a cleft palate. Thus is demonstrated the possibility of a cholesteatoma arising from stratified squamous epithelium which has not entered the middle ear via a perforation of the drum membrane. The author, however, believes the latter to be much the more frequent source of cholesteatoma. The case supports Tumarkin's contention. It also illustrates the possibility of the origin of squamous cell carcinoma from this site.

SUMMARY.

1. The embryonic development of the tube is briefly reviewed.

2. Evidence is presented to show that lumen of the infant tube is not larger than that of the adult.

3. An organized lymphoid nodule in the submucosa of the lumen of the Eustachian tube was found in only one ear in a study of 250 cases (500 ears).

4. A case of the lumen of the tube extending superiorly to a very thin tegmen at the isthmus is illustrated as an anomaly.

5. A case showing patches of stratified squamous epithelium in the bony tubes of an infant with a history of cleft palate is presented.

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50 West 59th Street.

DIAGNOSTIC PHASES OF MASTOIDITIS.*

DR. JOHN WINSTON FOWLKES, New York.

There are many well known and established phases of mastoiditis, but in addition there are others which present some of the most interesting and delicate problems with which the otologist of today has to deal. In this paper I shall deal especially with some of the phases which have some particular meaning and should be considered an integral part of the entire picture. To borrow a term from the electrical engineers, a physician must be equipped, at all times, to carry the maximum load. In other words, he should be able to visualize the process as a whole, with all of its possible complexities.

Since Hippocrates the danger and severity of ear infections have been thoroughly recognized. I am sure you are familiar with the quotation in which he said, "Acute pain in the ear, with continued strong fever, is to be dreaded, for there is danger that the man may become delirious and die." Since that time many facts have been learned and many aids developed to give us a truer picture of the real pathologic process.

Anatomy: From an anatomical standpoint certain features of the clinical as well as X-ray findings must be borne in mind.

1. In the very young the cells are normally more numerous, smaller, and present much thinner trabeculae than in the adult bone.

2. The cellular distribution may vary, the zygomatic cells sometimes being as numerous and extensive as the rest of the mastoid.

3. The size of the mastoid varies to a great extent, depending especially on whether there have been previous inflammatory attacks. As an example, the mastoid bone of an adult will be found to be smaller if there is a history of inflammatory attacks in childhood; also, in the mastoid which has been retarded in its growth we find the knee of the lateral sinus

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nearer the antrum than in cases without previous attacks. If this mastoid subsequently becomes infected, the sinus, from a surgical standpoint, is more vulnerable and the amount of discharge will be relatively less; the cortex becomes thicker and consequently the amount of pain elicited upon pressure will be correspondingly diminished (see Figs. 1, 2 and 3).

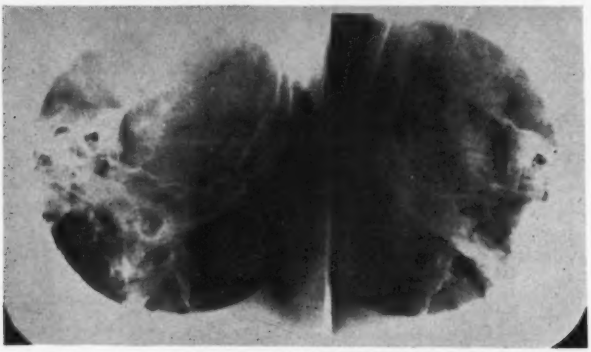

Fig. 1. Patient, age 22. Double chronic mastoiditis. Repeated attacks of inflammation in childhood. Illustrating the infantile type of mastoids.

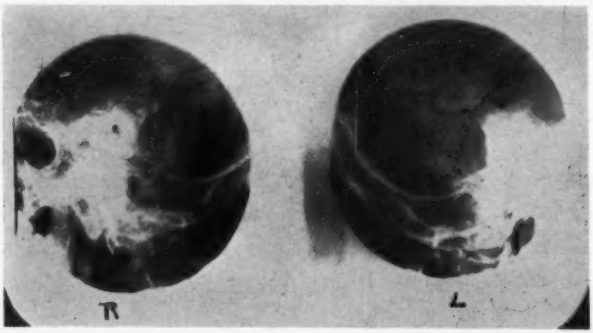

Fig. 2. Patient, age 43. (R) Normal in size and distribution of cells. (L) Discharge since childhood. Almost complete atresia of canal. Wassermann, four+. Chronic mastoiditis with infantile type of mastoid.

In addition to the ordinary position used in X-raying the mastoid to ascertain the amount of cloudiness and the amount of bone degeneration, there are two other positions which may be taken: The Mayer, which reveals any cavitation in the antral region, and the Stenvers showing the condition of the petrous apex. These two latter positions are especially

valuable if there is any question of chronicity; therefore, it is important to know the type of bone with which we are dealing (see Figs. 4, 5, 6 and 7).

Fig. 3. Patient, age 21. (R) Sclerotic mastoid. Repeated attacks since childhood. Now dry, drum intact. (L) Small sclerotic mastoid. Large perforation in drum, pusy discharge. Large cavitation in antral region at time of operation.

Fig. 4. Meyer position. Normal mastoid. No cavitation in antral region.

Bacteriology: When the mastoid becomes involved to any alarming extent, the bacteria found in the discharge from the middle ear are usually either streptococcus, pneumococ-

cus or staphylococcus, their order of frequency being as given above.

The streptococcus is more virulent; the pneumococcus, more insidious. The latter causes marked destruction, although there may be very slight signs and symptoms even when complications are at hand. I have seen cases of pneumococcus infection in which the middle ear had healed, the drum was normal in appearance and the hearing was also normal, but upon further investigation the mastoid was found to be entirely broken down. It is interesting to note that when dia-

Fig. 5. A. P. plate. Mastoiditis and petrositis, left side. Cavitation in left petrous apex. Pus under pressure at time of operation.

betes is a complicating factor in ear infections, the pneumococcus is generally the offending organism.

The staphylococcus when it has gained a foothold, even though less virulent, is prone to be more difficult to eradicate.

One must be ever aware of the type of organism to appreciate the behavior of the patient and the disease. Their clinical pictures may be entirely different, yet all may warrant a mastoidectomy.

Blood: The blood picture in obscure cases is usually of very little aid, since so often there is found only a mild leuko-

cytosis. If in repeated counts the percentage of the polymorphonuclear leukocytes increases, it should be considered a warning signal.

Temperature: A sudden rise in temperature with a chill is just cause for thinking immediately of a venous invasion (the lateral sinus); whereas, if the temperature remains high, meningitis is brought to mind. In both of these instances one should always be mindful of the possibility of complications such as a central pneumonia, acute bacterial endocarditis, pyelitis and nasal sinusitis.

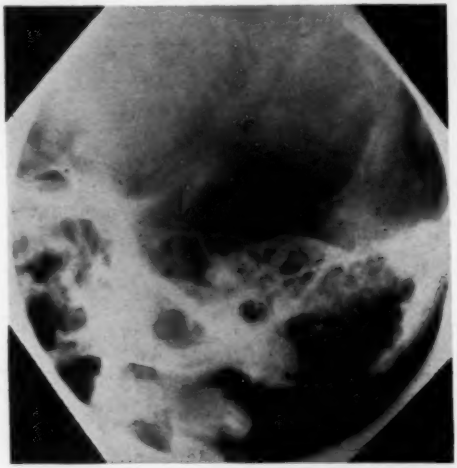

Fig. 6. Stenvers' position. Petrous apex clear.

There is usually very little temperature in uncomplicated cases after the initial rise in the very beginning of the infection; however, there is one particular type of case in which the temperature, although slight, is an indicator of great importance. This is one in which apparently nothing remains of the acute illness except a small amount of discharge and a rise in temperature once in 24 hours. In my opinion, in this type of case, although the patient seems quite well, it would be safer if a mastoidectomy were performed.

Pain and Tenderness: Pain is not always present, but there are certain types of pain which are of valuable diagnostic

significance and should be distinguished from the ordinary dull, intermittent discomfort associated with practically every case of otitis media.

1. A severe persistent pain in the antral region is diagnostic of an epidural abscess.

2. Soreness in the posterior muscles of the neck is often found upon careful examination. This is not to be confused with the board-like contraction found in meningitis and is probably due to the general uncomfortableness existing when the infection is progressing.

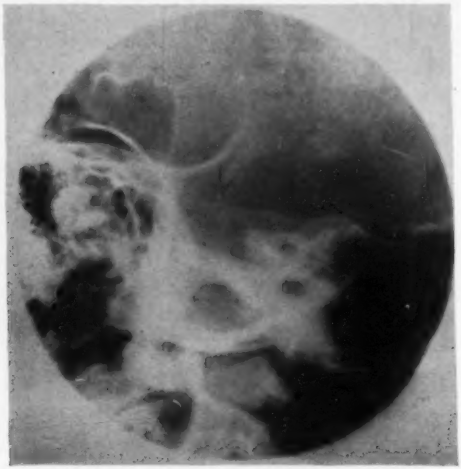

Fig. 7. Stenvers' position — opposite side of Fig. 6. Petrous apex cloudy. Moderate amount of pus at time of operation.

3. Pain in the lateral muscles of the neck, radiating to the shoulder, should not be attributed to an ear infection.

4. A boring-like pain which extends upward and radiates over the entire temporal bone immediately suggests the possibility of a petrous apex involvement. This is particularly true if it is accompanied by a VIth nerve paralysis and pain in the back of the eye on the same side.

5. A severe generalized headache in acute exacerbations of chronic mastoiditis may indicate the possibility of a brain abscess.

6. If the soreness and tenderness which had previously subsided recurs in a week or two, it should be regarded as an extension of the diseases and probably will require mastoidectomy; hence, it is of the utmost importance to note carefully the time, place, and type of pain and tenderness, as well as other otological findings.

Another phase in the diagnosis of mastoiditis has been produced since the introduction of the sulfa drugs. These drugs may cause a masking of mastoid symptomatology. For instance:

1. When these drugs are used, the temperature usually drops quickly, but later there may be a sharp rise accom-

Fig. 8. Fracture of longitudinal type, through squamous portion of temporal bone and base of the mastoid. Mastoiditis and brain abscess developed.

panied by a chill. This resembles an invasion of the blood stream by the infection and must be differentiated from a true septicemia.

2. The general symptoms are much less severe in character and the illness less acute.

3. After the drug has been administered for some time, the local signs may be misleading, as we may be unable to elicit any mastoid tenderness. The drum may be normal in appearance and the hearing normal, yet the mastoid may be entirely broken down by the infection. This has been aptly designated

as "silent mastoiditis" and is quite similar to the picture I have described in pneumococcus infections.

Fractures: Fractures of the base of the skull involving the mastoid with subsequent infection present interesting phases. These fractures take one of three courses: *1.* longitudinal; *2.* transverse; *3.* rupture of the petrous apex. The third type is usually fatal and will not be considered further.

1. Longitudinal: These fractures involve the tegmen tympani and run forward through the roof of the Eustachian

Fig. 9. Fracture demonstrable leading into mastoid. Complete loss of hearing of the involved ear.

tube. They may be linear or branches and usually do not involve the labyrinth; therefore, the usual signs and symptoms of internal ear involvement are not found. These fractures may extend posteriorly into the mastoid and rupture the lateral sinus. This will manifest itself by free bleeding into the middle ear and into the nasopharynx by way of the Eustachian tube (see Fig. 8).

2. Transverse: This type starts in the region of the jugular bulb or base of the mastoid, crosses the petrous pyramid and ends on the anterior surface of the petrous bone. In this

type the labyrinth and middle ear are more frequently involved, producing mastoiditis and meningitis, since the meninges are likely to be torn. The chances of such involvement are also augmented by the frequency of damage to blood vessels, the aqueductus vestibuli and aqueductus cochlear, usually resulting in total deafness. Such damage is usually the result of accidents and may be important from a medicolegal standpoint. It is always questionable just how much hearing, if any, will be regained and how long the expectant convales-

Fig. 10. All symptoms of a fractured skull. Complete loss of hearing in both ears. No fracture demonstrable in film.

cence will be; therefore, any statements regarding the hearing in such cases should be made with impunity (see Figs. 9 and 10).

The X-ray is an important aid in making the diagnosis and should be taken as soon as the condition of the patient permits, but it is not to be relied upon in all cases. In a large series of cases in Bellevue Hospital, in which the diagnosis was "fracture of the skull" and treated as such, no fracture was demonstrable in 25 per cent of the films.

Complications: Complications such as lateral sinus thrombosis, meningitis, brain abscess, petrositis and labyrinthitis are relatively rare. This especially is true if one has followed the case from the beginning and is allowed to choose the time of operation.

It must be remembered that osteomyelitis of the mastoid and surrounding bone may be very extensive before any alarming symptoms appear and when they do, prompt surgical interference is indicated.

Of course, operations are to be avoided but one of the most interesting characteristics of mastoid surgery is how well the patients respond, especially when one considers the amount of surgical work necessary to effect a prompt healing.

Where a bilateral mastoidectomy has been done and complications simulating blood stream infections ensue, the question arises as to which side should be investigated first. The following suggestions may be helpful in making the decision:

1. Neurological findings.

2. Pathology present at the time of the initial operations.

3. Question of tenderness over the large vessels of the neck.

4. Tobey-Ayer jugular compression test, bearing in mind the anomalies which may exist, as the left lateral sinus may be only a very small slit.

5. Whether there are differences in the eye grounds.

Although there is no one group of signs and symptoms which can be relied upon in all cases, there are many entities which, when considered collectively in their variations of severity, enable one to deduce important conclusions.

In addition to those considered above, it is equally important to evaluate other phases, such as skin, sleep, discharge, hearing, visual pathology of the drum and surrounding structures, and the general reaction of the patient to infection. Notwithstanding the fact that these may be considered familiar, it has been aptly said, "To regard whatever is common as of only inferior importance is an error to which all mankind is admittedly more or less liable."

Even with all of our modern laboratory help and clinical experience, man's keenest judgment, vision and intuition is often taxed to the utmost to satisfy his conscience in the passing of judgment on some cases.

12 East 63rd Street.

THE STATUS OF THE AUDITORY MECHANISM IN THE PILOT OF EXTENSIVE EXPERIENCE.

AN EVALUATION OF THE FACTORS CONTRIBUTING TO THE STATE OF HEARING DIMINUTION IN THE EXPERIENCED PILOT AND A CORRELATION OF THEM INTO OTOLOGICAL ENTITIES.

MAJ. CHARLES FIRESTONE, M.C., Lakeland, Fla.

In 1938, an article by this author, entitled "Bone Conduction in the Experienced Pilot and a Probable Interpretation," was published in THE LARYNGOSCOPE.[1] The conclusions expressed in this manuscript were arrived at from a study of bone conduction audiograms of 109 pilots, ranging in experience from 800 to 14,000 hours of flying. The opinion was expressed in this manuscript that these pilots, after varied periods of flying, had acquired a condition believed to be akin to otosclerosis. The latter opinion was expressed by the author notwithstanding the fact that these pilots invariably registered audiometric findings of a decreased perception of bone conduction, more or less proportional to the number of hours they had spent in the air. Supporting graphs from individual pilots and a composite graph arrived at from the supporting graphs were submitted to validate the conclusions expressed.

The 1938 *Yearbook of Eye, Ear, Nose and Throat*,[2] commenting on this manuscript, stated that it could not correlate the interpretation of otosclerosis in the presence of diminished bone conduction, inasmuch as the classical picture in otosclerosis presents an increase in bone conduction. Armstrong, in his volume, "The Principles and Practices of Aviation Medicine,"[3] echoes the criticism appearing in the *Yearbook*, citing this criticism in his text practically verbatim.

Studies of pilots' audiograms and observations made subsequent to the publication of the manuscript have not altered

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basically the conclusions expressed therein. The author, however, believes that while diminution of the perception of bone conduction is present, he considers the quantitative amount reported in the manuscript too high. Recency of flight was not considered in these recordings. Most of the pilots whose audiograms were reported were scheduled airline pilots who applied for examination in the interim between flights, when occupational trauma to the organ of Corti had been freshly incurred (vide infra). Subsequent observations showed that when experienced pilots were examined, following a rest from flying, the perception of bone conduction increased to an appreciable extent, but at no time to the vicinity of the normal level. The factor of determinism, *i.e.*, the genetic predispositional elements also produced variants in individual pilots. None of the pilots, however, exhibited normal perception of bone conduction. The author is convinced that a composite graph of bone conduction loss as measured in terms of perception is still valid and would revise only its degree of curvature.

Two to three years following the publication of the manuscript in THE LARYNGOSCOPE (loc. cit.), the author was able to obtain a pair of petrous pyramids of the temporal bones containing the entire middle and inner ear structures, from the remains of a pilot who had had over 5,000 hours of flying experience. These specimens were processed and sent to the Army Medical Museum for histological study. Col. James E. Ash,[4] curator of the museum, advised the author that the additional processing necessary before final histological study would be a lengthy one. After waiting for a period of one year, the histological study was completed and a report submitted to this author by Col. Ash.[5] Slide sections and a written interpretation of them are in my possession. The interpretation was stated by Col. Ash (loc. cit.) to have been obtained from various pathological laboratories of medical schools to which the slides had been dispatched for study. Otosclerotic changes were reported present in both these specimens. These findings were corroborated by the author's miscroscopic examination of the slides and by others who had examined them. It is realized that these findings represent the picture in an individual pilot and, of course, nothing even approaching a generalization can be made logically from these findings alone.

The pathological findings in idiopathic otosclerosis are generally stated to be confined to the osseous structure of the petrous pyramid and the stapedial-oval window articulation. Neither the terminal, intermediate, nor central portions of the neurologic components of the auditory apparatus are stated to be involved in the *basic* pathogenesis of this disease. Ballenger[6] states that degenerative and atrophic changes in the neurologic mechanism are present in this disease, when it is fully established. Inasmuch as the reports of pathologic specimens of otosclerotic cases are necessarily those of cases fully diagnosed and established, no absolute statement relative to a concomitant involvement of the neurologic element at the inception of its pathogenesis can be made. The author considers these degenerative changes of the neurologic component as changes that have supervened upon the basic pathology of the encasing osseous capsule; atrophic and vascular changes, supervening upon interruption in the normal chain of transmission of auditory stimuli, akin to disuse atrophy.

F. Koelsch,[7] writing in the September, 1935, issue of *Jahreskurse fur Arztiche Fortbildung*, finds certain structural changes in the bones and joints of workers with compressed air drills, and records a diminution of hearing in all of his cases, but fails to state which portion of the auditory mechanism is affected. The reaction of bony tissue to trauma (MacCallum,[8] Key and Conwell[9]) is stated to be a deposit of callus formation at the site of injury. Of course, the two latter authors imply gross localized direct traumata rather than generalized minute traumata. Koelsch (loc. cit.), however, deals more with the sort of traumata to which the aviator is exposed by the pounding vibrations of the machine he is flying.

Considered from a purely mechanical standpoint in keeping with principles of dynamics of sound, structural changes in the osseous system in general and the petrous pyramid in particular, coupled with the ankylotic changes in the stapedial oval window would, *per se*, produce changes in the coefficient of conductivity, analogous to the altered coefficient of conductivity found in metallic and nonmetallic substances whose proportions of composite elements have been changed by alloying, or whose densities have been altered by chemical treatment, such as tempering or aging. Considering Koelsch's (loc. cit.) findings, and MacCallum's (loc. cit.), and Key and

Conwell's (loc. cit.) statements relative to the response of bony tissue to trauma, the protective reaction of the osseous system to continued insult or injury would be that of otosclerotic changes in its structure; *i.e.*, callus formation. These alterations of callus deposits would be minute in size,—even to the point of being microscopic in places, and their distribution generalized throughout the osseous system, including the petrous pyramids. Under such conditions, the coefficient of conductivity of these structures must be expected to undergo changes.

When it is considered that the pilot is exposed to the multiple and cacophonous traumata from vibrations transmitted to him by the motors and propellers, to the extent that his entire anatomy actually physically vibrates in amplitudes and frequencies with which these plane motors and their appendages and superstructures vibrate, the exposure of the entire osseous system to these vibratory traumata should yield some degree of protective osseous changes described above, the degree approximately quantitatively proportional to the amount of time the pilot is exposed to these vibrations. At the same time we should keep in mind the determining factor; determinism, *i.e.*, in the language understood by the medico-pragmatically-minded, congenitally inherent quantitative variants in reaction of bony tissue to trauma. Topographical distributive factors would probably have to be considered. For example, the tubera ischii, the femora, the osseous structures entering into the formation of the feet and hands, and the coccygeal portion of the spinal column will be apt quantitatively to contain more callus deposits since they represent the primal contact points between the source of vibrations and the rest of the vibrating musculoskeletal system. [Koelsch (loc. cit.) in his study found the intensity of bony and articular changes to be somewhat directly proportional quantitatively to the proximity of the source of the vibrational trauma.] If this be true, then the findings of otosclerotic changes, in the specimens *(vide supra)* submitted to the U. S. Army Medical Museum, and studied under its direction, were to be expected and similar osseous changes in other pilots, the degree, depending somewhat directly upon the number of hours a pilot is exposed to these traumata (when the factor of determinism is disregarded), may also be expected. The author is of the opinion that such sclerotic changes in the entire osseus

system of pilots do exist, and arrives at the validity of this opinion from the expected reaction of osseous tissue to generalized trauma and from Koelsch's findings. The author further believes that the coefficient of conductivity of the osseous system under these conditions is altered and is altered in a positive direction; that is, it is increased because of an increase in density produced by the generalized deposit of callus in the bony skeleton.

If these latter hypotheses be valid, namely, that there is a generalized deposit of callus yielding changes in density and an increase in the coefficient of conductivity [and the work of Koelsch (loc. cit.) and the statements of MacCallum (loc. cit.), and Key and Conwell (loc. cit.), coupled with the otosclerotic changes found in the specimens submitted to the Army Medical Museum, validate them], the findings of decreased perception of bone conduction in experienced pilots would appear to be paradoxical. How, then, can a contradictory state of affairs of this sort be correlated and reconciled?

The paradox vanishes when the balance of the picture is viewed. The cacophony referred to supra has its clattering sonorous elements as well as its pure vibratory elements. These sonorous elements, *per se,* not considering for the moment the vibratory elements, produce changes in the terminal fibres of the organ of Corti (occupational deafness; *vide infra*), traumatizing it, and reducing its coefficient of *perceptive* acuity, to an extent that it is unable to perceive the impulses reaching it from a bony capsule of even increased conductivity. Thus the seeming paradox of actual physical increased bone conduction, with a diminution of perception of it, appears resolved.

In this connection, too, it should be apparent to the reader by this time that the otosclerotic condition referred to in this manuscript and in the manuscript published in 1938 (loc. cit.) is a mechanically induced otosclerosis, and is expected to differ from the idiopathic type of otosclerosis, in that the basic mechanical etiology of the osteo-otosclerotic condition is concurrently the etiologic factor in the genesis of the pathology in the neurologic element of the auditory apparatus. The nerve elements are undergoing pathologic changes simultaneously and, in part, from the same etiological trauma that produces the osteo-otosclerotic changes. This, in the opinion of

the author, is all-important and contains the kernel to a full understanding of the condition; *viz.*, while in the idiopathic otosclerosis the coefficient of bone conduction is increased, but, in the early stages, the perceptive nerve element is not altered. In the mechanical otosclerosis described above, the numerical *per se* coefficient of bone conductivity is increased, while at the same time the perceptive threshold of the organ of Corti is also increased. That is to say, an occupational nerve deafness akin to boilermaker deafness is produced at the same time that the bony changes are produced; hence, the presence of an absolute increase in the coefficient of bone conduction yields the readings of diminished bone conduction, because of the necessity, *in vivo*, of measuring bone conduction in terms of auditory perception. The bone conductive index is increased while the intensity of minimal stimuli has to be increased even greater to be perceived, since the nerve element had been traumatized both by musculoskeletal vibration and by clattering noises reaching it by air conduction.

At this juncture, the author desires to consider the neurologic elements of the auditory apparatus apart from the bony capsule encasing it. When it is considered that in flying the auditory nerve is exposed to the physical vibratory trauma transmitted to it from the continuous pounding of the motors, the propellers, and the entire ship and from uninterrupted noise and clatter transmitted to it via air waves, the diminution in hearing acuity sustained by the pilot falls into the category of an occupational deafness. Occupational deafness as an entity has been recognized for a long time. It has been found in boilermakers, in blacksmiths, etc. The latter occupational deafness however, lacks the element of continued concentrated vibrations in its etiological factorial. Until the advent of the concentrated use of the airplane, vibrational factors entering into the etiology of occupational deafness did not present themselves, although Koelsch *(vide supra)* reported diminished hearing in occupations embracing continuous vibratory traumata.

It is seen, therefore, that in the experienced pilot there is set up a train of events which yields both sclerotic changes combined with neurologic damage, and the entity "pilots' otosclerosis" would appear a valid one, as well as one that should be understood by the otologist, both from an interpretative

standpoint and from a socioindustrial standpoint, when, in the future, he is called upon to contribute to the adjudication of disability claims. In the opinion of this author, "pilots' deafness" as an occupational condition will be encountered, and the otologist's evaluation of it will be sought.

With the above as a background, a consideration of the pilots' stereotyped statement, when apprised of the diminution of their hearing, merits appraisal at this point. This author has had extensive experience in the examination and handling of pilots for a period of nine years. During this period, thousands of pilots have passed under his review and care. In almost every instance when a pilot who has had extensive flight experience is apprised of the audiometric findings in his case, he counters with the statement that he is able to detect the slightest motor dysfunction in flight, long before his junior co-piloting officer has recognized it; yet, these junior co-piloting officers, who have had little flying time, yield more nearly normal and, in some cases, normal, audiometric graphs. This author believes that the statements of these experienced pilots are not only true but are to be expected, inasmuch as the generalized osteosclerotic changes, the sclerotic changes in the otic bony capsule and the changes in the stapedial oval window articulation render these more experienced pilots subject to paracusis Willisi. They gain their "sense" of the state of their motors more by bone conduction reaching them via the vibratory route than by air conduction. The manifestations of motor dysfunctions are expected to be transmitted to the pilot through the vibratory sense by bone conduction, and these older pilots would be expected to detect these states of affairs more readily than the junior pilots who, because of lesser experience, lack the "sense" to appraise the information they receive by bone conduction from their skeletal systems, and who depend almost wholly on the auditory sense. The statements in the last sentence will be more lucid when the following is considered.

These experienced pilots appear to have developed a new composite sense to a high degree: composite of the kinesthetic, vibratory and the auditory, — the result of which is a consciousness of the state of performance of the apparatus that they fly, and with which they themselves vibrate in synchrony. An altered frequency or amplitude of vibration of

the plane, or any of its vibrating components, particularly of one or more of the motors, is detected by these pilots not only by means of their auditory apparatus but by means of the kinesthetic, vibratory and auditory senses combined. This may be termed "aviator's motor sense" and the degree of development of this sense is in a measure the coefficient of the status of efficiency of a given pilot.

The author offers the following experimentation carried out in multimotored planes in flight for a period of four hours, which leads him to believe that the auditory sense by air conduction alone during flight can be a detriment rather than an adjuvant to the attainment of this "aviator's sense." The pilot in each case was requested to dyssynchronize the motors of the plane so that the number of revolutions of one motor varied from appreciable down to minimal differences from the number of revolutions normal to the cruising speed of the plane. This was carried out unbeknown to the other officers and crew members in the plane. It was found invariably with 100 per cent of the crew members, as well as in 100 per cent of the number of trials, that the officers and crew members, including the author, were able to detect minimal dyssynchronies of the motors immediately when they plugged their external auditory canals. When the auditory canals were left patent, minimal dyssynchronies were not detected in some cases and in other cases their detection was slow and uncertain. It would be valid to conclude from these findings that the sensorium of the aviator gains more essential information from the perception of vibratory stimuli than it does from the perception of pure auditory stimuli, that perception of sound reaching the sensorium by air conduction tends to dilute the vibratory stimuli; and in the resulting perception of the cacophony there is confusion and less acute awareness of what is perceived.

An evaluation of these findings in conjunction with the remarks preceding them with a view to gleaning their significance and, if possible, their applicative potentialities, leads the author to the following considerations:

1. The "aviator's sense," being a composite as stated above, appears to have the vibratory as its most important component.

2. The vibratory sense, while it cannot be wholly divorced from the auditory sense, inasmuch as all vibration yields sound, is a sense perceived by the sensorium independent of the perception of its concomitant sound and represents a sense integer that has to be assigned a place in the sensorium somewhere between the kinesthetic and the auditory sense. In other words, the sensorium is able to detect an intrinsic vibratory sense even if it were possible to remove the sonorous element of vibration.

3. This sense differs from the kinesthetic sense inasmuch as the motion inherent in vibration is spatially infinitesimal. The spatial factor, for practical purposes, is nonexistent since the synchrony of the motion of the pilot with the machine annihilates relative spatial differences between them.

4. The occlusion of the external auditory canals, because of the resultant depollution of the vibratory stimuli from the air conduction stimuli, "purifies" the perception of the vibratory stimuli reaching the sensorium by bone conduction and enables the officers and crews of these planes to more readily detect dyssynchronies of the motors at their minimal threshold of dyssynchrony.

5. Auditory stimuli reaching the perceptive apparatus by means of air conduction are not only of negative importance but actually of a disservice to the orienting mechanism of the pilot in his relation to the apparatus he is flying.

6. The aviator unconsciously as well as consciously depends on his conductive vibratory sense more than on his perception by air conduction for information concerning the functioning of the motors and the structures to which they impart motive power.

Synthesizing the manuscript appearing in THE LARYNGOSCOPE (loc. cit.) with what has been stated above, it would appear —

1. That a mechanically induced osteo-otosclerosis resulting from prolonged trauma to the bony skeleton in general and to the otic bony capsule in particular may be considered a definite entity.

2. The mechanical etiological factors of vibration and noise yielding these changes in the bony capsule density simultaneously produce trauma to the neurologic portion of the auditory apparatus raising the threshold of perception.

3. The increased bone conduction present in this mechanically induced osteo-otosclerosis is physically present in the sense that the coefficient of conductivity is raised; however, the perceptive threshold of the organ of Corti has been raised simultaneously by the etiologic factors producing the former, and the increase in bone conduction is not perceived. The nerve suffers traumatic and vascular changes as in other occupational deafness; *i.e.,* boilermaker deafness.

4. We, therefore, correlate the existence of a true mechanically induced osteo-otosclerosis with an increased coefficient of bone conduction. Since bone conduction in the living can be measured relatively only in terms of perception, and since the perceptive elements of the pilot have been doubly traumatized, the resultant registration of *diminished* bone conduction, as measured in terms of perception, is understandable. In other words, prolonged flight over a period of years may be expected to produce generalized osteosclerotic changes and sclerotic changes in the otic bony capsule, with the introduction of a real increase of bone conductivity, but with a virtual register of diminished bone conduction when measured in terms of perception. The ratio of the increase of conductivity in this mechanically induced otosclerosis, omitting factors of determinism, would be expected to vary approximately inversely to the perception of it and may be expressed by the formula $C^x:C::R:R^x$,where C^x is the new index of conductivity and C is the normal index of conductivity of the bones of a given pilot, and where R is the normal index of receptivity and R^x represents the occupationally deafened organ of Corti.

5. The author is led to the recognition of a new entity; one of occupational otosclerosis with diminished *perception* of bone conduction coupled with diminution of hearing by air conduction, and a general dulling of the auditory sense because of the concomitant trauma to the neurologic portion of the auditory apparatus.

6. The vibratory sense of the pilot is of paramount importance to him in enabling him to detect readily motor failures in their incipiency. Occlusion of the external auditory canals prevents the noises of the motors and propellers from reaching his auditory apparatus by air conduction. This is a definite help to the pilot's desideratum.

CONCLUSIONS.

1. The author is led to restate[5] the conclusions expressed in THE LARYNGOSCOPE manuscript; that aero-otosclerosis or "pilot's otosclerosis" exists as an entity.

2. It is a mechanicaly induced osteo-otosclerosis coupled with diminished *perception* of bone conduction.

3. Audiometric findings in pilots who have had extensive flight experience show diminished perception of bone conduction.

4. So-called "pilot deafness" is both of a conductive and neurogenic type.

5. "Aviator's motor sense" is a composite, but is largely a vibration perception sense. This motor sense is rendered less acute by simultaneous perception of sound waves by air conduction.

RECOMMENDED APPLICATIONS.

1. That it be made mandatory on every pilot to obstruct fully his external canals during flight, not only to prevent trauma to the auditory apparatus but also to render the vibration perceiving mechanism more alert to incipient motor dyssynchronies.

2. That the armed forces alter their criteria of physical qualifications with respect to hearing acuity by air conduction, since hearing by air conduction (the whispered voice test) is not the paramount factor subjectively and objectively depended upon by the aviator in flight. Bone conduction should be as much, if not more, a criterion. Thus an applicant for flying who had had a middle ear disease during childhood and sustained certain adhesive sequellae in the middle ear which reduced his hearing by air conduction, say to 15/20

or even 10/20, and who has no other pathology, should be considered qualified for flying and in some cases of multimotored flying even be preferred.

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PRELIMINARY REPORT OF COMPLETE CLOSURE OF THE MASTOIDECTOMY WOUND BY PRIMARY SUTURE.*

DR. ARTHUR J. HERZIG, New York.

There is nothing original in closing a mastoidectomy wound completely as Blake, of Harvard, reported a series of cases in which he employed primary suture some 40 years ago, using the blood clot as a medium for filling the exenterated mastoid wound.

The author has used the complete closure by primary suture in several cases. He followed the suggestion of Dr. Blake in one case and in three cases he filled the wound with 1-200 acriviolet solution and in seven cases with equal parts of sulfanilamide and sulfathiazole powder.

Procedure employed was that of complete exenteration of all diseased bone; the sulfa powder was introduced into the wound, completely filling it (an average of two drams was used) and after making sure there was no oozing of blood, the periosteum was approximated with catgut sutures. (The author has been suturing the periosteum separately for over 30 years, previously permitting an opening in the lowermost portion for drainage. In all cases where this procedure was followed there were no wound depressions.) More sulfa powder was dusted on the sutured periosteum, and then the skin wound was closed with either Michel clips or silkworm gut; no drain whatsoever was used. More of the sulfa powder was placed on the skin wound and in the external auditory canal, the usual wick being introduced and a dressing applied.

All of these patients had frequent urinalyses as well as differential blood counts during their hospitalization. Following their discharge from the hospital they were observed at various periods for two months. All wounds closed by first intention. No dressings were applied after the fifth day, sutures having been removed on the fourth day. In all cases

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the aural discharge ceased on about the fourth day, the canals remaining dry.

It was observed that there was a slight leucopenia of about 20 per cent on or about the fourth day following the operation, this condition correcting itself within 48 hours. There was also a 10 per cent reduction in the red blood cell count, as well as an increased lymphocyte count on the fourth day, which corrected itself within 48 hours. These cases showed no toxic manifestations whatsoever. No sulfa crystals were found in the urine at any time.

The author in his final report will enumerate more in detail the comparison between the sulfa-filled wounds and those in which acriviolet 1-200 is used. The youngest of these cases was 5 months old and the oldest, 12 years. Since then a similar procedure has been employed in a much older case, the report of which will follow at a later date.

I am indebted to Dr. David Soloway (Resident, Department Otorhinolaryngology, Flower and Fifth Avenue Hospitals) for his valued assistance at these operations and in the preparation of the case histories, and thank him for his co-operation.

667 Madison Avenue.

REVIEW OF RECONSTRUCTIVE SURGERY OF THE FACE — 1942-1943.*

DR. FRANK MCDOWELL, St. Louis.

The literature on reconstructive surgery of the face from April, 1942, to April, 1943, has been small in amount and, in general, has reflected the trends of the times. This has been a period for consolidation, review and evaluation of previously developed procedures, though a few new and outstanding things have been reported. The arrival of foreign periodicals has dwindled to such an extent that their review at present is quite unsatisfactory.

FACIAL INJURIES.

Haynes[15] describes the treatment of wounds and deformities of the orbital region and makes several suggestions regarding the very difficult problem of the detached and displaced internal canthus. When possible, the internal palpebral ligament should be isolated and reanchored to the anterior lacrimal crest. If contracture of the lateral palpebral ligament has occurred, it may be necessary to sever it. It is occasionally feasible to reconstruct an internal ligament from fascia lata. For epiphora, he advises taking out the lacrimal gland, a procedure which is by no means universally accepted.

Kirby and Town[17] review the diagnosis and treatment of injuries of the eyes and lids, including burns, lacerations, ectropion, coloboma of the lid, etc. They have found that dacryocystorhinostomy is unsuitable for infected sacs following trauma because of the usual involvement of the ethmoids and displacement of local tissues, and excision of the sac has been the only satisfactory procedure. They also note, in agreement with many others, that no completely satisfactory operation for symblepharon has been devised.

Minsky[22] uses a special "intramarginal splinting suture" to prevent notching at the tarsal border after suturing vertical lacerations of the lids.

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Childrey[8] notes that most fractures of the malar bone are comminuted with involvement of the antral or orbital walls. He prefers a transantral approach through the canine fossa for most of them, packing up the orbital floor with iodoform gauze when it has been depressed.

Parker[25] reviews many well known procedures used for maxillary fractures and Williams[33] does the same with regard to the whole face.

Adams[1] has devised a new and promising technique for treatment of transverse maxillary fractures to hold the lower fragment up in place. A small hole is drilled through the anterior rim of the orbital floor and a stainless steel wire loop passed through it and down subcutaneously to connect with dental wires on teeth in the fragment. When the orbital floor is also split by a vertical fracture, it can be fixed directly with wires through drill holes before doing the above procedure, or the external angular process can be used for the superior point of fixation. Any method which helps in discarding the cumbersome extraoral appliances and tractions used so much in the last war would seem to be an advance, and as such this one is apt to get a good reception.

Brown and McDowell[4] have published a secondary report on their method of internal wire fixation for severe fractures of the lower jaw. Its use is not yet advised for simple fractures, but it seems to be one of the most direct and simple methods for fixation of edentulous jaws or fragments. A note on external bar fixation is included. The latter seems to be helpful as a space retainer when whole segments have been blown out or when comminution is extreme.

Hubinger[16] discusses the treatment of ununited fractures in the symphysis region. He uses acrylic splints for a long time and if union is not obtained, does an open operation.

Blair and Byars[3] emphasize the desirability of early proper treatment for face injuries and feel quite definitely that one should not wait for the swelling to subside before beginning treatment. A good resumé of the care of injuries in this region is given.

Several books have appeared in connection with facial injuries, including those by Fry, *et al.*,[13] Thoma,[32] Parker,[26]

Major,[19] and the official military manual.[23] The book by Fry, Shepherd, McLeod and Parfitt[13] was written in England and the methods have been evaluated in the light of their experiences in this war, which have not been inconsiderable. The description of the fundamental anatomy and pathology of the injuries is well done. Obsolete methods are largely omitted, or only mentioned as such. They are somewhat more enthusiastic about silver cast splints than most American surgeons, but perhaps we should explore the possibilities of this method more fully. The whole book is refreshing when compared to the usual books in this field. They describe so well the commonplace difficulties which beset everyone that the reader immediately realizes that this is not the work of any arm-chair surgeons.

Major[19] has written a good book on fractures of the jaws and facial bones, and Parker[26] has brought out a good synopsis in the same field. Thoma's[19] book is well done and deals with traumatic surgery of the jaws. All of them include the usual standard methods of treatment for these injuries, and some of them also have the new external bar fixation. They are all written primarily from the dental point-of-view, and certain sections, particularly those dealing with anatomy, burns, shock, infections, placing of sutures, etc., are apt to prove rather elementary to some surgeons.

The Manual of Standard Practice of Plastic and Maxillo-facial Surgery,[23] which was prepared by the subcommittee under the auspices of the National Research Council, appeared last year.

NOSES.

MacCollum[18] brings up the interesting possibility that the warping of rib cartilage transplants which sometimes occurs may be due to the fact that the cells lie predominantly in a curved line. In order to avoid this warping, he prefers the use of bone to build up noses, obtaining it from the iliac crest. When a columellar strut is desired, the periosteum on one side is left intact, and the end of the transplant is hinged down. Pelliciari[28] reviews the history and recent literature on fresh and preserved cartilage.

W. B. Davis[9] describes various plastic procedures on the nose, including straightening old fractures, cutting down

large noses, building up saddle noses with cartilage, cutting down rhinophymas, excision of carcinomas and flap repairs. Greeley[14] also discusses the repair of various nasal deformities together with the treatment of abrasions and accidental tattoos, keloids and scar contractures. Berson[2] introduces his own rhinometer, an instrument for measuring the glabellar and columellar-lip angles, not altogether dissimilar to the profilometer devised by Straith some years ago.

Parsons[27] immobilizes nasal fractures with plaster of Paris, a procedure seldom used by others.

Wolfe[34] advances the interesting theory that perhaps both rhinophyma and acne rosacea are due to an abnormal secretion of the androgenic hormones. He also reports an interesting case associated with an apparent vitamin C deficiency. After suitable preoperative preparation, he shaved the nose down in the usual way, but thought that cold compresses were better than hot ones for stopping the bleeding.

SKIN GRAFTING.

Byars[7] describes the indications for and the technique of doing full thickness skin grafts, illustrating the excellent results which may often be obtained by their use. He points out the objectionable heaviness of flap repairs about the chin, lip and neck, and the undesirable wrinkling that may occur in thinner free grafts, about the face and neck. In contradistinction to ideas expressed by some others, the article demonstrates that there are certain deformities which can be better repaired by a free full thickness graft than by any other method known at the present time.

Brown and McDowell[5] note that the thin scar epithelium that covers granulating wounds spontaneously is lacking in any dermal elements and, therefore, possesses only slight resistance to trauma. Both the thick split and full thickness skin grafts contain dermal pads and can be used to replace this thin scar epithelium with a stable surface. If the granulating area is of any considerable size, or if it is in an area that will be subject to considerable wear, much can be saved if the grafting is done immediately without waiting for spontaneous healing. In many instances, contractures can also be avoided. They discuss the emergency use of homografts

in badly debilitated patients and describe the processes concerned in their dissolution.

Stone[31] has published an excellent review of the knowledge to date concerning heteroplastic grafting of various tissues and organs with an outline of future possibilities.

Poth[29] has described two variations of split grafting, neither of which would seem to have much place in modern surgery. One method involves piecing tiny chips of split graft together on the back of a soap box with gauze and collodion and could very well be avoided by cutting a graft large enough in the first place. The other method consists of dicing up split grafts into small pinches and scattering them over the wounds. This is called "flagstone grafting" and that is very likely what the results resemble (others have called these "postage stamp grafts").

Brown and McDowell[6] have written a book on the skin grafting of burns. The indications for, and the methods of doing the various types of grafts are described. It is noted that many of these repairs can be better done and with greater facility by using free grafts rather than cumbersome pedicle flaps.

MISCELLANEOUS.

Newman[24] discusses the repair of prognathic and retruded jaws. In most instances, he prefers bilateral osteotomy of the rami above the nerve canals. For retruded chins, he also transplants derma with attached fat, feeling that the fat does not atrophy so readily when attached to derma and that it feels more natural than bone or cartilage.

Scher[30] describes three patients with associated chin and nose deformities. In one, he did a bilateral osteotomy of the rami for prognathism and an osteoplastic procedure on the nose. In another, he cut down a hump nose and transplanted preserved cartilage to a retruded chin. In the last one, a hump was removed from the nose and transplanted on to the retruded chin.

Dorrance and Loudenslager[12] point out a rather common condition to which little attention has been paid, calling it "hypermotility of the upper lip." It is characterized by a high upper lip which goes even higher on smiling, so that not

only the teeth but the alveolus is displayed. Their correction has consisted of going in through the buccal fornix on either side and dividing the levator muscles of the upper lip.

McCall and Gardiner[20] report three cases of facial paralysis following mastoid surgery. One was a physiological block with spontaneous recovery; another was due to depressed bone chips in the canal and cleared five months after decompression of the canal. The last one was first seen 18 months after operation and was repaired with a free nerve graft.

Dorrance and Bransfield[10] note that in covering an area of the skull in which the scalp and periosteum has been denuded, an adjacent scalp flap should be rotated over the defect and the resultant periosteal bed covered with a free skin graft.

Marino[21] has published an excellent book in Spanish on the cleft lip. The anatomy, embryology, pathogenesis and historical operations are included, as well as the various current methods of repair.

Dorrance and Bransfield[11] have written what may very well be a classical article on cleft palate surgery. They re-emphasize that many of the speech defects in these patients may be due to insufficient length of the palate. In the ordinary push-back or elongation operations which have been used in the past, the nasal surface of the flap that is set back is raw and may contract secondarily to partially mitigate the result. They show various methods of skin grafting the nasal surface of such flaps before setting them back, also methods of elongating complete single and double clefts with skin grafts on the nasal surface. A modification of Wardill's V-Y maneuver is included in the elongation of the complete clefts. The details are too complicated for review here, but anyone interested in palate surgery would do well to read the original article.

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